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Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites

Clement Associates, Inc., Fairfax, VA

Prepared for

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CLEMENT ASSOCIATES, INC.

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CLEMENT ASSOCIATES, INC.
1515 WILSON BOULEVARD
ARLINGTON, VA 22209

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The chemical profiles are intended to serve as a concise reference with information on the physicochemical properties, transport and fate, toxicity, and regulatory standars for individaul chemicals identified by the Office of Waste Program Enforcement (EPA) at hazardous waste sites. These profiles can be used in conjunction with the Toxicology and Endangerment Assessment Handbooks.

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CHEMICAL, PHYSICAL, AND BIOLOGICAL PROPERTIES OF COMPOUNDS PRESENT AT HAZARDOUS WASTE SITES

Final Report

Prepared for:

U.S. Environmental Protection Agency

Prepared by:

Clement Associates, Inc.
1515 Wilson Boulevard
Arlington, Virginia 22209

Under Subcontract to:

GCA Corporation
Bedford, Massachusetts 01730

September 27, 1985

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TO: U.S. EPA
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INTRODUCTION

Early in 1984, Clement Associates was requested under Subcontract No. 1-625-999-222-003 to GCA Corporation to provide support to the EPA Office of Waste Programs Enforcement (OWPE) in preparing a catalog of chemicals of concern at hazardous waste sites. Clement was given primary responsibility for two phases of this task. First, Clement was to assist GCA in developing an on-line catalog of chemicals present at hazardous waste sites. Second, Clement was to prepare profiles summarizing the chemical, physical, and biological properties of these chemicals. The chemical profiles are intended to serve as a concise reference with information on the physicochemical properties, transport and fate, toxicity, and regulatory standards for individual chemicals identified by OWPE at hazardous waste sites. They are not meant to be thorough, quantitative reviews and should not be used as substitutes for a good literature review on the characteristics of the specific chemicals of concern at a particular site.

During the first phase, Clement staff searched the consent decrees, administrative orders, and complaint files obtained from OWPE for the names of toxic chemicals detected at enforcement sites. They recorded the chemicals present at each site, the media in which the chemicals were detected, and the highest concentration of each chemical in a particular medium. This information was sent to GCA for inclusion in a computer data base. Also during this phase of the task, Clement developed dichotomous scores for each chemical indicating whether or not it had any of the following characteristics: carcinogenicity, reproductive toxicity/teratogenicity, mutagenicity, acute toxicity, chronic toxicity, toxicity to domestic animals, and toxicity to terrestrial and aquatic wildlife. The list of chemicals compiled and the criteria used for reaching these dichotomous determinations are included in Appendix A to this report.

During the second phase, Clement prepared profiles on the toxic chemicals that had been detected at hazardous waste sites. These profiles are based mainly on secondary sources, but the primary literature was consulted when necessary. Each chemical profile has five sections: Chemical and Physical Properties, Transport and Fate, Health Effects, Toxicity to Wildlife and Domestic Animals, and Regulations and Standards. A short summary; an introduction presenting the CAS registry number, chemical formula, IUPAC name, synonyms and trade names of the compound, and background information (where needed); and a reference list were also included.

These profiles can be used in conjunction with the Toxicology and Endangerment Assessment Handbooks. These handbooks present concepts and information that are important in understanding the health effects and other properties of pollutants identified at hazardous waste sites.

Brief descriptions of the various sections of the profiles and a general list of references are presented below.

Chemical and Physical Properties

Information on the following chemical and physical properties were obtained for each of the profiled compounds:

Atomic or Molecular Weight: The weight of an atom or molecule of a chemical expressed in atomic mass units. One atomic mass unit equals one-twelfth the mass of a carbon-12 atom.

Boiling Point: The temperature in degrees Celsius at which the vapor pressure of the compound is equal to or slightly greater than the atmospheric pressure [760 mm of mercury(Hg)].

Melting Point: The temperature in degrees Celsius at which a material changes from a solid state to a liquid state at atmospheric pressure.

Specific Gravity: The weight of a given volume of the compound at a specified temperature relative to the weight of an equal volume of water at 4 degrees Celsius.

Solubility in Water: The maximum amount of the chemical that will totally dissolve in water at a given temperature.

Solubility in Organics: The ability of the chemical to dissolve in specified organic compounds at a given temperature.

Log Octanol/Water Partition Coefficient: The log of the ratio of the amount of the chemical that will totally dissolve in n-octanol to the amount that will dissolve in water.

Vapor Pressure: The pressure (usually expressed in millimeters of mercury) exerted by the vapor phase of the chemical in equilibrium with the solid or liquid form at a given temperature.

Vapor Density: The weight of a given volume of the chemical relative to the weight of the same volume of air.

Henry's Law Constant: An expression of the distribution of the chemical between air and water at equilibrium. Usually defined as the ratio of the partial pressure of the compound in air measured in atmospheres to the mole fraction of the compound in a water solution.

pKa: A measure of the extent of dissociation of a material, which is defined as the pH at which half of the compound is ionized. pH is defined as the logarithm of the reciprocal of the concentration of hydrogen ions in a solution and ranges from 0 for the most acidic solution to 14 for the most basic.

Flash Point: The temperature at which a flammable liquid or solid gives off enough vapor to allow ignition of the vapor and air mixture.

Transport and Fate

The transport and fate of chemicals in the environment depends on the properties of both the chemical and the environmental medium in which it occurs. Because of the effects of the latter, the transport and fate of a compound can only be discussed in general terms when specific information on the characteristics of the environmental medium in which it is present is not available. Therefore, the transport and fate section in the profiles only provides general background information and will not apply in all cases. For a more thorough treatment of the principles governing chemical movement and fate in the environment, one of the general references listed at the end of this section should be consulted.

At any specific hazardous waste facility, the transport and fate of chemical contaminants must be assessed or modeled based on site-specific environmental information. Such assessment or modeling is often a complex task and requires expertise in a variety of scientific disciplines, including environmental chemistry and modeling.

Health Effects

The adverse health effects of greatest concern are those that cause death or are irreversible and seriously impair the normal functioning of the individual. Cancer is of concern because it is so often fatal and because of the broad agreement that there is no safe dose for many types of carcinogens. Mutagenicity, or genetic toxicity, is primarily important because an alteration in the genes of a cell may be the first step in tumor formation and may cause reproductive toxicity or teratogenicity. Reproductive toxicity decreases the individual's ability to produce viable young, while teratogenicity leads to the production of malformed offspring. Chronic toxicity involves effects that develop after long-term exposure (for several years) to a chemical. Acute toxicity refers to the effects that result from very short-term, usually single dose, exposure to a material. Subchronic exposure falls between chronic and acute exposure and usually involves exposure to a toxic agent for weeks or months. For chronic, subchronic, or acute toxicity, the effects of greatest concern are those that cause serious impairment and are irreversible. For a detailed description of these toxic effects, see the Toxicology Handbook or one of the general references listed at the end of this section.

For the purposes of these profiles, Clement scientists attempted to identify those effects of a particular chemical most likely to cause serious harm to exposed human populations. Because the profiles are primarily intended for use by EPA personnel dealing with hazardous waste sites, the toxic effects considered were those most likely to affect the two potentially exposed populations: on-site workers and people living near the site. For on-site workers who are exposed to site contaminants for short periods, the effects caused by acute or subchronic exposure are most important. For populations surrounding a site that are exposed to low levels of contamination for long periods, chronic effects are the greatest concern.

Toxicity to Wildlife and Domestic Animals

Toxic chemicals are a major concern as environmental contaminants if they cause either a potentially irreversible decline in one or more species or a decline in the aesthetics of an area. Toxicants can adversely affect a species by poisoning its members or by reducing their food supply. Poisoning can occur either by direct exposure in a contaminated area or, for predators or scavengers, by secondary exposure via contaminated prey or carrion. The latter method of exposure is of major concern because many persistent chemicals can be biomagnified to toxic levels. Furthermore, predators and scavengers near the top of the food chain are fewer in number than prey species and may also be less able to adjust to a declining population by increasing reproductive output. The classic example of this type of effect is the decline in predatory birds in the United States and Europe due to the ingestion of prey contaminated with the persistent organochlorine pesticides, such as DDT. In addition to species at the top of the food chain, endangered or threatened species are of major concern because even a small decline in the population of a species may be enough to cause its extinction.

Regulations and Standards

Several federal agencies and at least one private association have established recommended or mandatory maximum exposure levels for toxic chemicals. The U.S. Environmental Protection Agency (EPA) has developed health effects assessments (HEAs) for chemicals commonly detected at hazardous waste sites. These HEAs contain acceptable daily intake levels for subchronic and chronic exposure to noncarcinogens by either inhalation or oral routes of exposure and contain CAG unit risks for carcinogens. In addition, EPA has established the Interim Primary Drinking Water Standards, which specify the maximum levels of various chemicals allowable in water used for public consumption. EPA has also prepared criteria documents on 129 priority pollutants, which specify the maximum concentrations of these chemicals in ambient water at which the water can be regarded as acceptable for the protection of aquatic organisms and human health.

The Carcinogenesis Assessment Group (CAG) at EPA has performed carcinogenic risk assessments on many compounds. The "unit risks" (excess risk of cancer associated with lifetime exposure to 1 mg/kg/day of the chemical) calculated for these chemicals can be used to determine levels of exposure that are likely to have a low probability of causing cancer. A list of the unit risks developed by CAG and a brief description of this measure is included in Appendix B to this report.

Several agencies have developed allowable exposure levels for occupational exposure (40 hours per week) to airborne chemicals. The National Institute for Occupational Safety and Health (NIOSH) has reviewed the available data on numerous industrial materials and published criteria documents that contain recommended maximum levels of exposure to these materials in the workplace. The Occupational Safety and Health Administration (OSHA) has established regulations governing exposure to hazardous materials in the workplace. These standards differ from NIOSH recommendations in that they are mandatory. The American Conference of Governmental Industrial Hygienists (ACGIH), a nongovernmental association, has also recommended allowable exposure limits for workplace chemicals; many of these recommended limits, or Threshold Limit Values (TLVs), have been adopted as standards by OSHA.

The regulations, standards, and recommended exposure levels pertaining to each chemical have been included in the chemical profiles. These regulations were established using the best available scientific information, but they may change as improved data become available. EPA is currently finalizing the health effects assessments, proposing recommended maximum contaminant levels for drinking water, and developing health advisories for several of the chemicals presented in these profiles. Therefore, the values presented in the profiles may not reflect current scientific information in every case, although they will be generally applicable.

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CHEMICAL PROFILES

Chemical	CAS Number
Acenaphthene	83-32-9
Acenaphthylene	208-96-8
Acetic acid	64-19-7
Acetone	67-64-1
Acrolein	107-02-8
Acrylonitrile	107-13-1
Aldrin/Dieldrin	309-00-2 60-57-1
Alkanes	
Alkyl benzenes	
Anthracene	120-12-7
Antimony	7440-36-0
Arsenic	7440-38-2
Asbestos	1332-21-4
Barium	7440-39-3
Benzene	71-43-2
Benzidine	92-87-5
Benzo(a)anthracene	56-55-3
Benzothiazole	95-16-9
Beryllium	7440-41-7
Butanol	71-36-3
Cadmium	7440-43-9
Carbon tetrachloride	56-23-5
Chlordane	57-74-9

Chemical	CAS Number
Chlorine	7782-50-5
Chlorobenzene	108-90-7
Chlorobenzilate	510-15-6
Chloroethane	75-00-3
bis(2-Chloroethoxy)ethane	112-26-5
bis(2-Chloroethyl)ether	111-44-4
Chloroform	67-66-3
p-Chloro-m-cresol	59-50-7
1-Chloro-3-nitrobenzene	121-73-3
Chromium	7440-47-3
Chrysene	218-01-9
Cobalt	7440-48-4
Copper	7440-50-8
Cresols	1319-77-3
Cyanide	57-12-5
Cyanuric acid	108-80-5
DDT	50-29-3
Dibromochloropropane	96-12-8
Dichlorobenzenes	
1,1-Dichloroethane	75-34-3
1,2-Dichloroethane	107-06-2
1,1-Dichloroethylene	75-35-4
1,2-trans-Dichloroethylene	156-60-5
2,4-Dichlorophenol	120-83-2
2,4-Dichlorophenoxyacetic acid	94-75-7

Chemical	CAS Number
1,2-Dichloropropane	78-87-5
1,3-Dichloropropene	542-75-6
Dicofol	115-32-2
Diethyl phthalate	84-66-2
Diisobutyl ketone	108-83-8
Dimethylaminoethyl methacrylate	2439-35-2
Dimethylaniline	121-69-7
Dimethylnitrosamine	62-75-9
2,4-Dimethylphenol	105-67-9
n-Dioctyl phthalate	117-84-0
1,4-Dioxane	123-91-1
Diphenylethane	1103-29-7
Endrin	72-20-8
Ethanol	64-17-5
Ethanolamine	141-43-5
Ethyl acetate	141-78-6
Ethylbenzene	100-41-4
Ethylene/diethylene glycol	107-21-1 111-216-6
Ethyl ether	60-29-7
Ethyl hexanediol	94-96-2
bis(2-Ethylhexyl)phthalate	117-81-7
Fluoranthene	206-44-0
Formaldehyde	50-00-0
Heptachlor	76-44-8

Chemical	CAS Number
Hexachlorobenzene	118-74-1
Hexachlorobutadiene	87-68-3
Hexachlorocyclohexane	608-73-1
Hexachloroethane	67-72-1
Hexachlorophene	70-30-4
Hexane	110-54-3
Iron	7439-89-6
Isobutyl alcohol	78-83-1
Isopropyl ether	108-20-3
Lead	7439-92-1
Lithium	7439-93-2
Magnesium	7439-95-4
Manganese	7439-96-5
Mercury	7439-97-6
Methacrylic acid	79-41-4
Methanol	67-56-1
Methyl chloride	74-87-3
Methylene chloride	75-09-2
Methyl ethyl ketone	78-93-3
Methyl isobutyl ketone	108-10-1
Methyl parathion	298-00-0
Naphthalene	91-20-3
Nickel	7440-02-0
Nitrocellulose	9004-70-0
Nitrophenol	

Chemical	CAS Number
Pentachlorophenol	87-86-5
Phenanthrene	85-01-8
Phenol	108-95-2
Phenyl ether	101-84-8
Phosphoric acid	7664-38-2
Phosphorus (white)	7723-14-0
Picric acid	88-89-1
Polychlorinated biphenyls	1336-36-3
Polychlorinated dibenzo-p-dioxins	
Polycyclic aromatic hydrocarbons	
Selenium	7782-49-2
Silver	7440-22-4
Sodium	7440-23-5
Sodium chlorate	7775-09-9
Stoddard solvent	8052-41-3
1,2,4,5-Tetrachlorobenzene	95-94-3
2,3,7,8-Tetrachloro-dibenzo-p-dioxin	1746-01-6
1,1,2,2-Tetrachloroethane	79-34-5
Tetrachloroethylene	127-18-4
Tetraethyl lead	78-00-2
Tetrahydrofuran	109-99-9
Thallium	7440-28-0
Titanium	7440-32-6
Toluene	108-88-3

Chemical	CAS Number
Toxaphene	8001-35-2
Trichlorobenzenes	
2,3,6-Trichlorobenzoic acid	50-31-7
1,1,1-Trichloroethane	71-55-6
1,1,2-Trichloroethane	79-00-5
Trichloroethylene	79-01-6
Trichlorofluoromethane	75-69-4
2,4,5-Trichlorophenol	95-95-4
2,4,5-Trichlorophenoxyacetic acid	93-76-5
2,4,5-Trichlorophenoxy propionic acid	93-72-1
tris(2,3-Dibromopropyl)phosphate	126-72-7
Vanadium	7440-62-2
Vinyl chloride	75-01-4
Xylenes	1330-20-7
Zinc	7440-66-6

ACENAPHTHENE

Summary

Acenaphthene is a two-ringed polycyclic aromatic hydrocarbon (PAH). Although little specific information on acenaphthene is available, information on related PAHs suggests that acenaphthene is not very persistent in the environment and that biodegradation is the ultimate fate process. Acenaphthene has not been shown to be carcinogenic or mutagenic, but it does cause liver and kidney damage at high exposure levels.

CAS Number: 83-32-9

Chemical Formula: $C_{12}H_{10}$

IUPAC Name: Acenaphthene

Chemical and Physical Properties

Molecular Weight: 154.21

Boiling Point: 279°C

Melting Point: 96.2°C

Specific Gravity: 1.225 at 0°C

Solubility in Water: 3.42 mg/liter at 25°C

Solubility in Organics: Soluble in ethanol, toluene, chloroform, benzene, and acetic acid

Log Octanol/Water Partition Coefficient: 4.33

Vapor Pressure: Less than 0.02 mm Hg at 20°C

Vapor Density: 5.32

Transport and Fate

Acenaphthene, like other polycyclic aromatic hydrocarbons (PAHs), can be emitted into the environment by both natural and anthropogenic sources. Since very little information is available on this compound specifically, its environmental fate is largely inferred from data for PAHs in general. In air, acenaphthene can be transported as adsorbed matter on suspended particulates. Ambient air samples collected in Sydney, Australia, contained 0.07 μg/100 m^3, indicating that atmospheric transport occurs and that individuals in urban environments may be exposed to measureable levels.

In surface water, direct, rapid photolysis of dissolved acenaphthene may be an important water-related environmental fate. It is probable that singlet oxygen is the oxidant and that the reaction products are quinones. Volatilization may play a role in acenaphthene transport, depending on mixing rates in both the water and air columns. However, adsorption to sediments is probably the dominant aquatic transport process. Consideration of the log octanol/water partition coefficient for acenaphthene and of the behavior of other PAHs indicates that acenaphthene can be strongly adsorbed onto suspended and sedimentary particulate matter, especially particulates high in organic content.

Based on information concerning related compounds, it is likely that bioaccumulation of acenaphthene is short term, especially for vertebrates. Although it is rapidly accumulated after exposure, it also is rapidly metabolized and excreted. Consequently, bioaccumulation is not considered an important fate process. Biodegradation is considered the ultimate fate process for acenaphthene. Based on information for related compounds, it is probable that acenaphthene is readily degraded by microbes. Biodegradation is likely to be more rapid in the soil than in aquatic systems. However, studies indicate that biodegradation may be more important in those aquatic systems that are chronically affected by PAH contamination.

Health Effects

Negative results are reported for a test of acenaphthene carcinogenicity based upon neoplastic induction in the newt *Triturus cristatus*, but the reliability of the test system for predicting mammalian carcinogenicity is not established. Other carcinogenicity studies involving exposure to acenaphthene as one component of complex mixtures of PAHs and other substances report both positive and negative results. However, the relative importance of individual components in the mixtures tested cannot be determined, and no conclusions involving acenaphthene can be drawn. Studies using several different bacterial test systems provide no evidence of mutagenicity. No information concerning its teratogenicity or reproductive toxicity is available.

The most thoroughly investigated effect of acenaphthene is its ability to produce nuclear and cytological changes in a variety of microbial and plant species. Most of these changes, such as increases in cell size and DNA content, are associated with a disruption of the spindle mechanism during mitosis and the resulting induction of polyploidy. However, there is no known correlation between these effects and the biological impact of acenaphthene on mammalian cells.

Very little is known about the human toxicity of acenaphthene. It has been shown to be irritating to the skin and mucous membranes and to cause vomiting if swallowed in large quantities.

In both rats and mice, subchronic oral exposure causes loss of body weight, changes in peripheral blood, increased aminotransferase levels in blood serum, and mild morphological damage to the liver and kidneys. The oral LD_{50} is 10 g/kg for rats and 2.1 g/kg for mice. Kidney and liver damage is greater after subchronic exposure to acenaphthene than after acute exposure.

Toxicity to Wildlife and Domestic Animals

In acute toxicity tests for freshwater organisms, EC_{50} values of 41,200 and 1,700 µg/liter are reported for the cladoceran *Daphnia magna* and the bluegill, respectively. In saltwater species, 96-hour LC_{50} concentrations for the mysid shrimp and the sheepshead minnow are 970 and 2,230 µg/liter, respectively. A chronic value of 710 µg/liter is reported for the sheepshead minnow, and the acute-chronic ratio for this species is 3.1. No other aquatic life chronic data are available. The freshwater alga *Selenastrum capricornutum* and the saltwater alga *Skeletonema costatum* are both relatively sensitive to acenaphthene exposure, with 96-hour EC_{50} values for chlorophyll a and cell number of approximately 525 µg/liter and 500 µg/liter, respectively.

The steady state bioconcentration factor for acenaphthene in the bluegill is 387, with a tissue half-life of less than 1 day. By using the bluegill data and an adjustment factor to allow for differences in lipid content, the bioconcentration factor for acenaphthene and the edible portions of all freshwater and estuarine aquatic organisms consumed by Americans is estimated to be 242. Reports of acenaphthene in foods is limited. One study reports levels of 3.2 µg/kg (the detection limit) or greater in the tissues of shellfish of an unspecified species and location.

A study summarizing the toxicity of a variety of compounds to wild and domestic bird species indicates that the LD_{50} of acenaphthene for the redwinged blackbird is greater than 100 mg/kg.

Furthermore, the study reports that acenaphthene did not significantly deter feeding by the blackbird even when it was present in food at relatively high concentrations.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are inadequate for establishing final criteria. EPA did report the lowest valves known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 1,700 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 970 μg/liter
Chronic toxicity: 710 μg/liter

Human Health

The available data are inadequate for establishing a human health criterion.

Organoleptic criterion: 20 μg/liter

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ACENAPHTHYLENE

Summary

Acenaphthylene is one of the polycyclic aromatic compounds (PAHs) and is produced from both anthropogenic and natural sources. It is moderately persistent in the environment; photolysis in water and biodegradation in soil are the most likely fate processes. No information is available on the carcinogenicity, reproductive toxicity, or general toxicity of acenaphthylene. However, it exhibited weak mutagenic activity in a microbial test system, and like other PAHs, it probably is a skin irritant.

CAS Number: 208-96-8

Chemical Formula: $C_{12}H_8$

IUPAC Name: Acenaphthylene

Chemical and Physical Properties

Molecular Weight: 152.21

Boiling Point: 265 to 275°C

Melting Point: 92°C

Specific Gravity: 0.8988 at 16°C

Solubility in Water: 3.93 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, ether, and benzene

Log Octanol/Water Partition Coefficient: 4.07 (calculated value)

Vapor Pressure: 10^{-3} to 10^{-2} mm Hg at 20°C (estimate based on data for structurally similar compounds)

Transport and Fate

Environmental transport and fate is largely inferred from data for polycyclic aromatic hydrocarbons (PAHs) in general, because specific information for acenaphthylene is lacking. Rapid, direct photolysis of acenaphthylene to quinones may be an important process in surface waters. Oxidation is probably too slow to be a significant environmental process. However, data for some PAHs suggest that oxidation by chlorine or ozone may be a significant fate process when these oxidants are available in sufficient quantities. Volatilization may play a role in acenaphthylene transport depending on mixing rates in both the water column and air column. For acenaphthylene, it is probable that adsorption generally is the most important aquatic transport process. Consideration of its log octanol/water partition coefficient and of the behavior of other PAHs indicate that acenaphthylene can be strongly adsorbed onto suspended and sedimentary particulate matter, especially particulates high in organic content. It is likely that this compound can be readily transported as adsorbed matter or suspended particulates in air or water.

Based on information concerning related compounds, it is likely that bioaccumulation of acenaphthylene is short term, especially for vertebrates. Although PAHs are rapidly accumulated, they also are rapidly metabolized and excreted, and consequently bioaccumulation is not considered an important fate process. PAHs can be metabolized by multicellular organisms and degraded by microbes. Degradation by mammals is likely to be incomplete, with parent compound and the metabolites being excreted by the urinary system. Biodegradation by microorganisms is probably the ultimate fate process for acenaphthylene. Biodegradation generally appears to be more efficient in soil than in aquatic systems. However, experimental data indicate that biodegradation may be more important in those aquatic systems that are chronically affected by PAHs contamination.

Atmospheric transport of PAHs can occur, and these materials can be returned to aquatic and terrestrial systems by wet and dry deposition. Some PAHs can enter surface and groundwaters by leaching from polluted soils.

Health Effects

There are no epidemiological or case studies suggesting that acenaphthylene is carcinogenic in humans. There are no reports of carcinogenic, teratogenic, or reproductive effects in experimental animals. Acenaphthylene is reported to have weak mutagenic activity in a Salmonella typhimurium test system (Kaden et al. 1979).

No information concerning acute or chronic toxicity is available. Like many other PAHs, acenaphthylene may be a skin irritant, but little specific information is available.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity to domestic animals and wildlife are not available. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 119.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

The available data are inadequate for determining the cancer risk for acenaphthylene specifically. Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

Risk	Concentration
10^{-5}	28 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

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ACETIC ACID

Summary

Acetic acid is a relatively weak acid with a pKa of 4.7. It is soluble in water. Acetic acid irritates the skin, eyes, and mucous membranes, and it may have adverse reproductive effects at high dose levels. Acetic acid vapors are known to form explosive mixtures and toxic fumes when combined with air.

CAS Number: 64-19-17

Chemical Formula: CH_3COOH

IUPAC Name: Acetic acid

Important Synonyms and Trade Names: Glacial acetic acid, vinegar acid, ethylic acid, ethanoic acid, and methanecarboxylic acid

Chemical and Physical Properties

Molecular Weight: 60.05

Boiling Point: 118°C

Melting Point: 16.6°C

Specific Gravity: 1.05 at 20°C

Solubility in Water: Very soluble

Solubility in Organics: Soluble in alcohol, acetone, benzene, glycerin, ether, and carbon tetrachloride

Vapor Pressure: 11.4 mm Hg at 20°C

Vapor Density: 2.07

Flash Point: 40°C (closed cup)

Transport and Fate

No information was available on the transport and fate of acetic acid. However, some generalizations can be made based on chemical and physical properties. Acetic acid is

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extremely soluble in water and therefore is probably transported in surface and groundwater. Only a small amount is likely to volatilize from natural surface water owing to its high solubility in water.

Health Effects

Acetic acid is not considered to be a carcinogen. Mutagenic effects were observed in an assay using the microorganism *Escherichia coli*. Sex chromosome loss and nondisjunction were reported in *Drosophila melanogaster*. Oral administration of 700 mg/kg to pregnant females produced behavioral effects in newborn rats.

Acute toxicity depends on the chemical form; the free acid is more toxic than the salt. Irritant effects on the human gastrointestinal tract were seen at concentrations as low as 1,470 µg/kg after oral administration. Inhalation produced irritant effects at an exposure level of 100 mg/m^3 administered for 1 hour, while severe toxic effects were associated with exposure to 500 mg/m^3 for the same period. The irritation caused by acetic acid usually affects the skin, eyes, mucous membranes, or the exposed teeth. Irritant effects on humans and animals do not appear to be cumulative. The LD$_{50}$ in the rat given neutralized acetic acid orally was 3,310 mg/kg, while the LD$_{50}$ value in mice following intravenous administration was 525 mg/kg.

Five workers exposed to high concentrations (196-490 mg/m^3 at peak concentrations) for 7 to 12 years, experienced short-term loss of sensitivity, conjunctivitis, bronchitis, pharyngitis, and erosion of exposed teeth. Ingestion of concentrations between 0.01% and 0.25% (approximately 10-160 mg/kg) had no toxic effects when administered to rats over a 2- to 4-month period. When concentrations of 0.5%, corresponding to daily doses of about 330 mg/kg, were administered, an immediate and progressive decrease in food consumption and growth was observed.

Acetic acid vapors are known to form explosive mixtures and toxic fumes when combined with air.

Toxicity [illegible] Wildlife and Domestic Animals

Only limited information on the toxicity of acetic acid to aquatic organisms is available. Acute exposure of bluegills and goldfish resulted in a 96-hour EC$_{50}$ of 75 mg/liter and 100 mg/liter, respectively. The LC$_{50}$ for shrimp ranged from 100 to 330 mg/liter when they were exposed for a 48-hour period in aerated water. The perturbation level in the protozoa (*Vorticella campanula*) was 12 mg/liter. In bacteria (*Pseudomonas* putida), cell multiplication was inhibited at 2,850 mg/liter, while 90 mg/liter inhibited multiplication for the algae (Microcystis aeruginosa).

No information on the toxicity of acetic acid to terrestrial wildlife or domestic animals was found in the literature reviewed.

Regulations and Standards

OSHA Standard: 25 mg/m^3 TWA

ACGIH Threshold Limit Values: 25 mg/m^3 TWA
37 mg/m^3 STEL

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ACETONE

Summary

Acetone is a commonly used solvent, which probably is not very persistent in the environment. It is considered to have rather low toxicity, and no chronic health hazards have been associated with exposure to it. Acetone is not very toxic to aquatic organisms.

CAS Number: 67-64-1

Chemical Formula: CH_3-CO-CH_3

IUPAC Name: Propanone

Important Synonyms and Trade Names: Dimethyl ketone, 2-propanone

Chemical and Physical Properties

Molecular Weight: 58.08

Boiling Point: 56.2°C

Melting Point: -95°C

Specific Gravity: 0.7899 at 20°C

Solubility in Water: miscible

Solubility in Organics: Soluble in alcohol, ether, benzene, and chloroform

Log Octanol/Water Partition Coefficient: -0.24

Vapor Pressure: 190 mm Hg at 20°C

Vapor Density: 2.00

Flash Point: -16°C (closed cup)

Transport and Fate

Very limited information on the transport and fate of acetone was found in the literature reviewed. However, ketones in general are probably not very persistent. Acetone has a high vapor pressure and therefore would be expected to volatilize readily, but because of its high water solubility, volatilization is probably limited. Once in the atmosphere, it is apparently

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oxidized. Acetone has a low octanol/water partition coefficient and therefore is probably not readily adsorbed. Biodegradation is probably important in determining the fate of acetone in the environment because of its aliphatic nature. Evidence of this is provided by the biological oxygen demand value, which was 72% of the theoretical value after 20 days at 20°C.

Health Effects

Acetone has not been tested in a carcinogenicity bioassay but gave negative results in a skin painting test and was not mutagenic in the Ames assay. No studies on animals for teratogenicity or reproductive toxicity have been done, but acetone was negative in a chicken egg injection study for teratogenicity.

Acetone is generally regarded as having low toxicity and therefore has not been extensively studied. Prolonged inhalation of high concentrations may produce irritation of the respiratory tract, coughing, headache, drowsiness, incoordination, and in severe cases, coma.

In animal studies, rats consuming doses of 18 mg/kg/day for 4 months showed reduced food consumption and growth. In behavioral studies, rats exposed to 6,000 ppm (14,200 mg/m^3) acetone for 4 hours/day, 5 days/week for 2 weeks showed modified avoidance and escape behavior after one exposure, but no changes after subsequent exposures. At 16,000 ppm (37,800 mg/m^3), altered responses were noted throughout the 2-week exposure period. No chronic health hazards have been associated with exposure to acetone.

Toxicity to Wildlife and Domestic Animals

The toxicity of acetone to aquatic organisms is low. The LC$_{50}$ value for sunfish was reported to be 14.2 g/liter, and the threshold concentration for immobilization of *Daphnia magna* was reported to be over 9 g/liter (McKee and Wolf 1963).

No information on the toxicity of acetone to terrestrial wildlife or domestic animals was found in the literature reviewed.

Regulations and Standards

NIOSH Recommended Standard (air): 250 ppm (593 mg/m^3) TWA

ACGIH Threshold Limit Values: 750 ppm (1,780 mg/m^3) TWA
1,000 ppm (2,375 mg/m^3) STEL

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AMERICAN INDUSTRIAL HYGIENE ASSOCIATION (AIHA). 1980. Hygienic Guide Series: Acetone

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ACROLEIN

Summary

Acrolein is an aldehyde that has been used as as aquatic herbicide. It is hydrated and then biodegraded in water and probably is not very persistent in the environment. Acrolein is mutagenic and may have toxic reproductive effects. It is a powerful irritant and can cause permanent lung damage when inhaled.

CAS Number: 107-02-8

Chemical Formula: CH_2CHCHO

IUPAC Name: 2-Propenal

Important Synonyms and Trade Names: Acrylic aldehyde; allylaldehyde, 2-propen-1-one, prop-2-en-1-al, acrylaldehyde

Chemical and Physical Properties

Molecular Weight: 56.1

Boiling Point: 52.5°C

Melting Point: -86.9°C

Specific Gravity: 0.8410 at 20°C

Solubility in Water: 200 g/liter

Solubility in Organics: Soluble in ethanol, ether, and acetone

Log Octanol/Water Partition Coefficient: -0.090

Vapor Pressure: 220 mm Hg at 20°C

Vapor Density: 1.94

Flash Point: -26.1°C

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Transport and Fate

Hydration to beta-hydroxypropionaldehyde, followed by biodegradation, is probably the most important aquatic fate for acrolein. The half-life for these processes is reported to be less than 4 days. Although volatilization can occur, its relative importance as an environmental process is not known. No information on the photolysis of acrolein in aquatic systems is available, but this process may proceed slowly in the atmosphere. Some oxidation of acrolein may occur in aquatic systems and in the atmosphere. The relatively high water solubility and the low log octanol/partition coefficient of acrolein make sorption and bioaccumulation unlikely environmental processes.

Health Effects

There is no unequivocal evidence to suggest that acrolein is carcinogenic in humans or experimental animals. There are no reports of teratogenicity, but intravenous administration during gestation is reported to increase postimplantation mortality. Acrolein also produces mutagenic effects in a variety of test systems.

Most reports of acrolein toxicity are associated with inhalation exposure. Acrolein is a powerful lachrymogen, and it is irritating to the eyes and to the mucous membranes of the respiratory tract. Irritant effects are observed in individuals exposed to 2.5 mg/m^3 or less. Higher concentrations can cause persistent lung damage. Exposure to approximately 350 mg/m^3 is reported to be fatal in humans within 10 minutes.

Lacrimation and irritation of the eyes and respiratory tract are the most commonly observed effects in experimental animals exposed to acrolein by inhalation. This compound also is reported to produce severe skin irritation. The inhalation and oral LD_{50} values in rats are 300 mg/m^3 and 46 mg/kg, respectively.

Toxicity to Wildlife and Domestic Animals

Available information indicates that acute and chronic toxicity [illegible] freshwater aquatic life occurs at concentrations as low as [illegible] and 21 µg/liter, respectively. Acute toxicity to saltwater aquatic life is reported to occur at concentrations as low as 55 µg/liter. Toxicity would occur at lower concentrations among species more sensitive than those tested.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 320 μg/liter

OSHA Standard: 0.25 mg/m^3

ACGIH Threshold Limit Values: 0.25 mg/m^3 TWA
0.8 mg/m^3 STEL

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ACRYLONITRILE

Summary

Acrylonitrile is an important chemical intermediate used in the plastics industry. It is fairly soluble in water and is also quite volatile. Photooxidation in the atmosphere and biodegradation are probably important fates in the environment. IARC has classified acrylonitrile as a suspected human carcinogen. It causes lung tumors in humans and animals exposed by inhalation and tumors at other sites in experimental animals exposed orally. Acrylonitrile is mutagenic and teratogenic, and it can damage the central nervous system, liver and kidneys.

CAS Number: 107-13-1

Chemical Formula: CH_2CHCN

IUPAC Name: 2-Propenenitrile

Important Synonyms and Trade names: Vinyl cyanide, cyanoethylene, properenitrile

Chemical and Physical Properties

Molecular Weight: 53.06

Boiling Point: 77.5 to 77.9°C

Melting Point: -83.55°C

Specific Gravity: 0.8060 at 20°C

Solubility in Water: 73,500 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, acetone, and benzene

Log Octanol/Water Partition Coefficient: -0.14

Vapor Pressure: 80 mm Hg at 20°C

Vapor Density: 1.83

Flash Point: 0°C

Transport and Fate

Direct photolysis of acrylonitrile in aquatic environments is unlikely. However, it may react with some naturally occurring aromatic rings in the presence of photosensitizing plant pigments or industrially produced dyes. Such conditions might occur in highly polluted surface waters or at a waste site. Volatilization is a major environmental transport process for acrylonitrile. Acrylonitrile can be volatilized from aquatic and terrestrial systems, and transported in the atmosphere as a vapor or adsorbed to particulates. Although acrylonitile can return to aquatic and terrestrial systems in precipitation, photooxidation in the troposphere is a significant environmental fate process. Hydrolysis and sorption probably are not important fate processes under natural conditions.

Bioaccumulation of acrylonitrile is not expected to be a significant process, but the cyanoethylation of proteins in aquatic biota may occur. Acrylonitrile is biodegraded by sewage sludge, but there may be an insufficient population of microorganisms in the water column and insufficient contact time for biodegradation to be effective in surface waters. In terrestrial mammals, acrylonitrile can be metabolized to thiocyanate and eliminated in the urine. Considerable species and organ differences in mammals' ability to detoxify acrylonitrile have been observed.

Health Effects

The International Agency for Research on Cancer (IARC) classifies acrylonitrile as a suspected human carcinogen. Epidemiologic studies of persons occupationally exposed to this compound revealed an excess of cancer at a number of sites, but particularly in the lung. In a two-year study with acrylonitrile incorporated in the drinking water of rats, increased incidences of subcutaneous tumors in the mammary region, Zymbal gland carcinomas, central nervous system astrocytomas, and squamous cell papillomas of the forestomach were observed in animals receiving doses as low as 35 ppm (approximately 8.75 mg/kg/day). Acrylonitrile also appears to be carcinogenic in experimental animals exposed by inhalation; it produces lung tumors in these animals. Acrylonitrile is mutagenic in a variety of test systems. It also produces maternal toxicity and teratogenicity in rats after oral or inhalation exposure, and in hamsters after intraperitoneal administration.

Acrylonitrile is readily absorbed from the respiratory and gastrointestinal tracts, and through the intact skin. Several studies report toxic effects of acrylonitrile due to occupational and accidental exposures. These results must be interpreted with caution, however, because the exposures

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Acrylonitrile is readily absorbed from the respiratory and gastrointestinal tracts, and through the intact skin. Several studies report toxic effects of acrylonitrile due to occupational and accidental exposures. These results must be interpreted with caution, however, because the exposures are often to a combination of substances. Some of the subjective complaints reported are headache, fatigue, vertigo, nausea, weakness, and insomnia. The clinical symptoms observed include anemia, jaundice, conjunctivitis, mild liver injury, abnormal blood and urine values, and functional disorders of the cardiovascular, hemopoietic, and central nervous systems. Contact allergic dermatitis, toxic epidermal necrosis, disturbed immunological reactivity, and sensitization have also been seen. Impaired pulmonary function and mortality have been reported in cases of acute exposure to very high concentrations of acrylonitrile.

Acrylonitrile, administered by various routes, can cause edema in the body organs of mice, rats, and guinea pigs, as well as damage to the central nervous system, liver, and kidneys. An oral LD$_{50}$ of 82 mg/kg is reported for rats. Rats and rabbits that inhaled 250 mg/m^3 (114 ppm) of acrylonitrile for 6 months exhibited changes in peripheral blood patterns; functional disorders in the respiratory, cardiovascular, and renal systems; and neuronal lesions in the central nervous system. Some of the same changes were observed at concentrations of 50 mg/m^3 (23 ppm). An inhalation LC$_{LO}$ of 500 ppm (approximately 1,100 mg/m^3) for 4 hours is reported for rats.

Toxicity to Wildlife and Domestic Animals

A 48-hour EC$_{50}$ of 7,550 µg/liter is reported for the freshwater invertebrate *Daphnia magna*. Among freshwater fish, 96-hour LC$_{50}$ values are 11,800 µg/liter for the bluegill, 18,100 µg/liter for the fathead minnow, and 33,500 µg/liter for the guppy. A 30-day LC$_{50}$ value of 2,600 µg/liter is reported for the fathead minnow. Data obtained during this study suggest that acrylonitrile has a definite chronic or cumulative effect and that adverse effects can be expected to occur at concentrations below 2,600 µg/liter in fish exposed for more than 30 days.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of acrylonitrile in water are:

Risk	Concentration
10^{-5}	0.58 μg/liter
10^{-6}	0.058 μg/liter
10^{-7}	0.006 μg/liter

CAG Unit Risk (USEPA): 0.24 $(\text{mg/kg/day})^{-1}$

NIOSH Recommended Standard: 4 ppm Ceiling Level

OSHA Standard: 2 ppm TWA (cancer hazard)
10 ppm (15-min Ceiling Level)

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ALDRIN/DIELDRIN

Summary

Aldrin degrades to dieldrin, which is very persistent in the environment. Both pesticides are carcinogenic in rats and mice and are teratogenic and reproductive toxicants. Aldrin and dieldrin cause liver toxicity and central nervous system abnormalities following chronic exposure. Both are also acutely toxic, with oral LD_{50} values of about 50 mg/kg. Both pesticides are very toxic to aquatic organisms and have been associated with large-scale kills of terrestrial wildlife in treated areas.

Background Information

Dieldrin is the 6,7-epoxide of aldrin and is readily obtained from aldrin under normal environmental conditions and by metabolism in animals.

CAS Number: Aldrin: 309-00-2
Dieldrin: 60-57-1

Chemical Formula: Aldrin: $C_{12}H_8Cl_6$
Dieldrin: $C_{12}H_8Cl_6O$

IUPAC Name: Aldrin: 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4:5,8-exo-dimethanonaphthalene

Dieldrin: 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-endo,exo-1,4:5,8-dimethanonaphthalene

Chemical and Physical Properties

Molecular Weight: Aldrin: 365
Dieldrin: 381

Melting Point: Aldrin: 104°C
Dieldrin: 176°C

Solubility in Water: Aldrin: 20 µg/liter at 25°C
Dieldrin: 200 µg/liter at 25°C

Solubility in Organics: Soluble in most organic solvents

Log Octanol/Water Partition Coefficient: No data found; probably greater than 5 for both chemicals

Vapor Pressure: Aldrin: 2.31×10^{-5} mm Hg at 20°C
Dieldrin: 2.8×10^{-6} mm Hg at 20°C

Transport and Fate

Aldrin evaporates rapidly from aquatic environments and also probably from soil. Photolysis probably occurs in the atmosphere after volatilization. Adsorption, especially by organic materials, is also an important fate process for this chemical. Aldrin is bioconcentrated by aquatic organisms by a factor of 10^3 to 10^4. Biotransformation by aquatic organisms and biodegradation are also important fate processes.

The primary product of aldrin degradation is its epoxide, dieldrin. Photolysis of aldrin also produces small amounts of photoaldrin, photodieldrin, and a polymerization product. Dieldrin is considered to be at least as toxic as aldrin and is quite persistent in the environment. Therefore, transformation of aldrin represents only a change of state and not detoxification of the chemical.

Dieldrin is one of the most persistent of the chlorinated hydrocarbons. Volatilization and possibly subsequent photolysis to photodieldrin are important transport and fate processes from surface water and probably from soil. Adsorption to sediments, especially organic materials, and bioaccumulation are also important in removing dieldrin from water. Biotransformation and biodegradation of dieldrin occur very slowly but may be the final fate processes in sediment.

Health Effects

Both aldrin ~~and~~ dieldrin are carcinogens, causing increases in a variety of tumors in rats at low but not at high doses and producing a higher incidence of liver tumors in mice. The reason for this reversed dose-response relationship is unclear. Neither appears to be mutagenic when tested in a number of systems. Aldrin and dieldrin are both toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation; while teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Chronic effects attributed to aldrin and dieldrin include liver toxicity and central nervous system abnormalities. Both chemicals are acutely toxic; the oral LD$_{50}$ is around 50 mg/kg, and the dermal LD$_{50}$ is about 100 mg/kg.

Toxicity to Wildlife and Domestic Animals

Aldrin and dieldrin are both acutely toxic to freshwater species at low concentrations. Tests in fish showed that the two chemicals had similar toxicities, with LC$_{50}$ values ranging from 1 to 46 µg/liter for different species. Final acute values for freshwater species were determined to be 2.5 µg/liter for dieldrin and 3.0 µg/liter for aldrin. Saltwater species were also quite sensitive to aldrin and dieldrin. The range of LC$_{50}$ values was similar to that for freshwater species: 2 to 100 µg/liter for aldrin and 1 to 34 µg/liter for dieldrin. The saltwater Final Acute Values were 1.3 µg/liter for aldrin and 0.71 µg/liter for dieldrin.

Chronic studies have been conducted on the effects of dieldrin on freshwater and saltwater species. For freshwater organisms, chronic values as low as 0.2 µg/liter were obtained. The Final Acute Chronic Ratio was determined to be 8.5, and the calculated Freshwater Final Chronic Value was 0.29 µg/liter. Only one chronic study was done on saltwater species. Therefore, the saltwater Final Chronic Value of 0.084 µg/liter was determined by dividing the Final Acute Value by the acute-chronic ratio. No chronic studies were performed on aldrin, but because its acute toxicity is comparable to that of dieldrin and because it is readily converted to dieldrin in animals and in the environment, it probably has similar chronic toxicity.

Both pesticides, and especially dieldrin, have been associated with large-scale bird and mammal kills in treated areas. Experimental feeding studies have shown that the chemicals are quite toxic to terrestrial wildlife and domestic animals at low levels.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: Aldrin: 3.0 µg/liter
Dieldrin: 2.5 µg/liter

Chronic toxicity: Aldrin: No available data
Dieldrin: 0.0019 μg/liter

Saltwater

Acute toxicity: Aldrin: 1.3 μg/liter
Dieldrin: 0.71 μg/liter

Chronic toxicity: Aldrin: No available data
Dieldrin: 0.0019 μg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations in water are:

Risk	Aldrin Concentration	Dieldrin Concentration
10^{-5}	0.74 ng/liter	0.71 ng/liter
10^{-6}	0.074 ng/liter	0.071 ng/liter
10^{-7}	0.0074 ng/liter	0.0071 ng/liter

CAG Unite Risk (USEPA): Aldrin: 11.4 $(\text{mg/kg/day})^{-1}$
Dieldrin: 30.4 $(\text{mg/kg/day})^{-1}$

ACGIH Threshold Limit Value:* 0.25 mg/m^3 TWA
0.75 mg/m^3 STEL

OSHA Standard (air):* 250 $\mu\text{g/m}^3$ TWA

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ALKANES

Summary

The high molecular weight alkanes (C_6 and greater) are major constituents of petroleum. Their transport and fate in the environment and their toxicity depend on both their chain length and branching. In general, the high molecular weight alkanes are rather persistent in the natural environment and biodegradation is probably an important fate process. These long chain alkanes generally are not very toxic, but they are irritants and several may be neurotoxic. Concentrations of 100 mg/liter of pentane, hexane, or heptane were not toxic to young coho salmon, but they did cause irritation.

CAS Number:

Chemical Formula: C_nH_{2n+2}

Chemical and Physical Properties

Molecular Weight: 14 x number of carbons + 2

Boiling Point: Increases with increasing molecular weight

Melting Point: Variable, depending on whether there is an even or an odd number of carbons and on branching (-91 to 20°C)

Specific Gravity: Increases with increasing molecular weight (0.7 to 0.8 at 20°C)

Solubility in Water: Essentially insoluble in water; solubility decreases with increasing molecular weight

Solubility in Organics: Soluble in benzene, carbon tetrachloride, chloroform, and other alkanes

Log Octanol/Water Partition Coefficient: Approximately 0.5 multiplied by the number of carbons in the range of [illegible] to [illegible] and increasing more slowly thereafter (calcu

Vapor Pressure: Decreases with increasing molecular weight

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Transport and Fate

Only limited information on the transport and fate of alkanes with high molecular weights (i.e., C_8 and greater) was found in the literature reviewed. However, some estimates of likely transport and fate processes can be determined from information on shorter chain alkanes, the chemical and physical properties of the specific compounds, and some limited data on biodegradation.

Alkanes are relatively nonreactive compounds and therefore are probably rather persistent in the environment. Branching tends to increase stability so that the alkane degrades even more slowly. The alkanes with the higher molecular weights have low vapor pressures and probably are not very volatile. However, their low solubility in aqueous media and consequent high level of activity may make them at least somewhat volatile. Once in the atmosphere, and in the presence of NO_x, slow photooxidation to alkanones will occur. Alkanes with more than six carbons have fairly high log octanol/water partition coefficients and probably bind readily to organic materials in soil and sediment. They are unlikely to be very motile in the environment. The principal fate of alkanes in aquatic and terrestrial environments is probably biodegradation by soil and aquatic microorganisms (Haines and Alexander 1974). These microorganisms can convert the alkanes to long-chain carboxylic acids such as oleic acid (Merdinger and Merdinger 1970).

Health Effects

No information was found suggesting that the alkanes are likely to be carcinogenic or mutagenic, or to cause reproductive toxicity. n-Hexane has been associated with polyneuropathy in chronically exposed workers and has induced peripheral nerve damage in animal studies. However, there is no evidence that longer chain or branched alkanes have this same effect. In tests examining suppression of the action potential in nerve cells, longer chain alkanes were progressively less active than shorter chain alkanes (Haydon et al. 1977). In LD_{100} studies on 13 C_5 to C_{17} alkanes injected into mice, Jeppsson (1975) noted that toxicity was generally positively correlated with the number of carbons up to C_8 (n-octane), after which toxicity usually declined. The alkanes are also irritants, with the effect becoming more severe as the number of carbons increases, at least up to C_9 (the heaviest alkane tested).

Toxicity to Wildlife and Domestic Animals

Morrow (1974) reported that pentane, hexane, and heptane at levels of 100 mg/liter in aerated seawater were not lethal to young Coho salmon but did cause irritation. No deaths occurred in mosquito fish exposed for 96 hours to n-heptane at a nominal concentration of 5,600 mg/liter in aerated, turbid water. (The actual concentration of alkane in these experiments may have been lower because of their insolubility, binding to particulates, and volatilization). A 24-hour LD_{50} value of 4 mg/liter was reported for goldfish exposed to either n-hexane or n-heptane. No other information on the toxicity of alkanes to wildlife or domestic animals was found in the literature reviewed.

Regulations and Standards

NIOSH Recommended Standard: 360 mg/m^3 TWA

OSHA Standard: 1,450 mg/m^3 TWA (octane)

ACGIH Threshold Limit Values: 1,450 mg/m^3 TWA (octane)
1,800 mg/m^3 STEL (octane)

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ALKYL BENZENES

Summary

Alkyl benzenes are a class of compounds that have a single aromatic ring with one or more aliphatic chains attached. They are not very persistent in the environment. Alkyl benzenes are not very toxic, but at high doses they are irritants and can cause central nervous system anomalies. They are generally toxic to fish at concentrations greater than 4 mg/liter.

Introduction

Several individual alkyl benzenes, specifically toluene, xylene, and ethylbenzene, are discussed in separate profiles. Because the information on other alkyl benzenes is rather limited and these compounds are likely to behave rather similarly, they are considered together in a general profile. Information on specific chemicals is provided in the attached table.

Chemical Formula: $C_6H_x(CH)_x$

Chemical and Physical Properties

Boiling Point: Directly related to number of carbons--100-190°C

Melting Point: Variable, generally around 25°C

Specific Gravity: Approximately 0.9 at 20°C

Solubility in Water: Inversely related to number of carbons:
C_8--approximately 160 mg/liter
C_9--approximately 70 mg/liter
C_{10}--approximately 15 mg/liter

Solubility in Organics: Soluble in acetone, benzene, and ether

Log Octanol/Water Partition Coefficient: Directly related to number of carbons.
C_8--approximately 3
C_9--approximately 3.5
C_{10}--approximately [illegible]

Vapor Pressure: Inversely related to number of carbons.
C_8--approximately 8 mm Hg
C_9--approximately 3 mm Hg
C_{10}--approximately 1 mm Hg

Transport and Fate

Alkyl benzenes will volatilize into the atmosphere from both the soil and surface water. Once in the air, they are attacked by hydroxyl radicals to form aldehydes, hydrooxalkylbenzenes, and nitroalkylbenzenes; they are also oxidized, and the chief product is peroxyacetylnitrate (PAN). The alkyl benzenes have log octanol/water partition coefficients of 3 to 4 and therefore are probably adsorbed by organics in soil and sediments. They are also biodegraded by soil and aquatic microorganisms. Alkyl benzenes probably are not very persistent in the environment.

Health Effects

The information on the potential health effects associated with exposure to the alkyl benzenes with 9 or more carbons is extremely limited. No data were available on the carcinogenicity of the alkyl benzenes. The results of mutagenicity assays on both ethylbenzene and xylene were negative. Ethylbenzene and xylene, and presumably the larger alkyl benzenes, are not teratogenic, but they do retard growth somewhat. The primary effects associated with exposure to the alkyl benzenes are narcosis, central nervous system anomalies, and irritation, particularly of the mucous membranes. Oral LD_{50} values in the rat for the larger alkyl benzenes are: ethyltoluene--5,000 mg/kg; isopropyl benzene--1,400 mg/kg; trimethylbenzene--5,000 mg/kg; diethylbenzene--5,000 mg/kg; and tetramethylbenzene--6,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

Very limited information was available on the toxicity of alkyl benzenes to wildlife, and none was found on their toxicity to domestic animals. In fish, 96-hour LC_{50} values were generally in the range of 10 to 100 mg/liter. Invertebrate species were apparently affected at similar concentrations. However, sensitive species were stressed at concentrations as low as 4 mg/liter, and young fry were often adversely affected at still lower concentrations. These studies were conducted on toluene, ethylbenzene, and xylene, and not on the longer chain or more substituted alkybenzenes. It appears that, in general, toxicity increases with increases in the number of attached alkyl groups and in chain length. However, these results did not hold in all studies. It is possible that decreased solubility and increased adsorption to particles would effectively limit the toxicity of larger alkyl benzenes to aquatic organisms.

Regulations and Standards

OSHA Standard (skin): 245 mg/m^3 TWA (isopropyl benzene)

ACGIH Threshold Limit Values (skin):

Isopropyl benzene:	245 mg/m^3	TWA
	365 mg/m^3	STEL
Trimethyl benzene:	125 mg/m^3	TWA
	170 mg/m^3	STEL

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ANTHRACENE

Summary

Anthracene is a three-ringed polycyclic aromatic hydrocarbon (PAH). It is probably moderately stable in the environment. Anthracene causes dermatitis and other skin disorders in humans.

CAS Number: 120-12-7

Chemical Formula: $C_{14}H_{10}$

IUPAC Name: Anthracene

Important Synonyms and Trade Names: Paranaphthalene

Chemical and Physical Properties

Molecular Weight: 178.22

Boiling Point: 340 to 355°C

Melting Point: 217°C

Specific Gravity: 1.24 at 27°C

Solubility in Water: 0.073 mg/liter at 25°C

Solubility in Organics: Soluble in acetone and benzene

Log Octanol/Water Partition Coefficient: 4.45

Vapor Pressure: 1.95×10^{-4} mm Hg at 20°C

Vapor Density: 6.15

Transport and Fate

Much of the information concerning transport and fate is inferred from data for polycyclic aromatic hydrocarbons (PAHs) in general, because specific information for anthracene is lacking. Rapid, direct photolysis of anthracene to quinones may occur in aqueous solution. Oxidation is probably too slow to be a significant environmental process. The available data suggest that volatilization may be a significant transport

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photodynamic response concentration of 0.1 μg/liter is reported for the freshwater protozoan Paramecium caudatum. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 478.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data are not adequate for establishing criteria.

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ANTIMONY

Summary

Antimony production has been associated with an increase in lung cancer in exposed workers. An inhalation study using rats yielded suggestive evidence that antimony trioxide causes lung and liver tumors, and several antimony compounds were mutagenic when tested using bacterial test systems. Female workers exposed to antimony compounds had an increased incidence of gynecological disorders and spontaneous abortions; similar effects were observed in an animal study. Antimony also causes cardiovascular changes in humans and may damage the myocardia.

Background Information

Antimony exists in a variety of chemical forms. It is found in any of four valence states (-3, 0, +3, or +5). In the environment, stibnite (Sb_2S_3) is the most common naturally occurring form of antimony, although it is also found as the native metal, as antimonides of heavy metals, and as antimony oxides.

CAS Number: 7440-36-0

Chemical Formula: Sb

IUPAC Name: Antimony

Chemical and Physical Properties (Metal)

Atomic Weight: 121.75

Boiling Point: 1750°C

Melting Point: 630.74°C

Specific Gravity: 6.684 at 25°C

Solubility in Water: Insoluble; some compounds are soluble

Transport and Fate

Antimony is present as the soluble oxide or antimonite (+3) salt in most natural waters. In reducing environments, volatile stibine (SbH_3) may be formed. Stibine is a gas at room temperature, and it is quite soluble in water. However, it is not stable in aerobic waters or air and is oxidized to form Sb_2O_3. The formation of stibine in bed sediments, which usually provide a reducing environment, may offer a mechanism for remobilization of antimony previously removed from solution. Biomethylation processes resulting in the formation of volatile stibine derivatives may also cause some remobilization of antimony. The extent to which sorption reduces the aqueous transport of antimony is unknown, but it is clear that sorption to clays and minerals is normally the most important mechanism resulting in the removal of antimony from solution. There also is a possibility that heavy metals in solution could react with antimonite or antimonate (+5) to form insoluble compounds. The importance of such processes is unknown, but it is likely that most species of antimony in natural waters are soluble and quite mobile and are eventually transported in solution to the oceans. Bioaccumulation appears to be only a minor fate process for antimony. Airborne transport of antimony in the form of particulates can also occur.

Health Effects

Antimony production has been associated with an increase in lung cancer among exposed workers, and one inhalation study in rats also indicated that antimony trioxide might produce lung and liver tumors. Several studies in bacterial test systems report that various antimony compounds, including antimony trioxide, antimony trichloride, and antimony pentachloride, may be mutagenic. Reports of effects on reproduction are limited. Among the effects on reproduction reported for humans are impairments to the female reproductive system. Female workers exposed to metallic antimony dust, antimony trioxide, and antimony pentoxide had an increased incidence of gynecological disorders and late spontaneous abortions. Antimony was found in the breast milk, placental tissue, amniotic fluid, and blood of the umbilical cord in exposed workers. Decreased weight gain was observed in children born of workers exposed to antimony. The same paper reports a study in which intraperitoneal administration of antimony produced changes in rats that support the findings of human reproductive effects.

Cardiovascular changes associated with exposure to antimony represent a serious health effect. Exposure to either trivalent or pentavalent antimonial compounds can produce electrocardiogram (ECG) changes in humans. Histopathological evidence of cardiac edema, myocardial fibrosis, and other signs of myocardial structural damage indicates that antimony may produce even more severe, possibly permanent myocardial damage in humans. Parallel findings of functional changes in ECG patterns and of histopathological evidence of myocardial structural damage have also been obtained in animal toxicity studies. Pneumoconiosis in response to inhalation exposure and dermatitis in response to skin exposure have also been observed among individuals exposed to antimony or its compounds.

Toxicity to Wildlife and Domestic Animals

Tests with antimony potassium tartrate and antimony trichloride in *Daphnia magna* reveal no difference in the toxicity of these two compounds. The LC$_{50}$ and EC$_{50}$ values for *Daphnia magna* and the fathead minnow, both freshwater species, range from 9,000 to 21,900 µg/liter. Chronic values for the fathead minnow and *Daphnia magna* are 1,600 and 5,400 µg/liter, respectively. Acute-chronic ratios for the fathead minnow and *Daphnia magna* are 14 and 3.5, respectively. The freshwater alga *Selenastrum capricornutum* is more sensitive than the animal species tested, with a 96-hour EC$_{50}$ of 610 µg/liter for inhibition of the synthesis of chlorophyll *a*. No detectable bioconcentration of antimony by the bluegill was observed. No definitive data concerning the toxicity of antimony to saltwater species or to other wildlife or domestic animals are available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 9,000 µg/liter
Chronic toxicity: 1,600 µg/liter

Saltwater

Acute toxicity: No available data
Chronic toxicity: No available data

Human Health

Criterion: 146 µg/liter

NIOSH Recommended Standard: 0.5 mg/m^3 TWA (antimony and all antimony compounds except stibine)

OSHA Standard: 0.5 mg/m^3 TWA

ACGIH Threshold Limit Value: 0.5 mg/m^3 TWA (antimony and its compounds as Sb)

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ARSENIC

Summary

Arsenic is a metal that is present in the environment as a constituent of organic and inorganic compounds; it also occurs in a number of valence states. Arsenic is generally rather mobile in the natural environment, with the degree of mobility dependent on its chemical form and the properties of the surrounding medium. Arsenic is a human carcinogen; it causes skin tumors when it is ingested and lung tumors when it is inhaled. Arsenic compounds are teratogenic and have adverse reproductive effects in animals. Chronic exposure to arsenic is associated with polyneuropathy and skin lesions. It is acutely toxic to some early life stages of aquatic organisms at levels as low as 40 μg/liter.

Background Information

Arsenic can be found in the environment in any of four valence states (-3, 0, +3, and +5) depending on the pH, Eh, and other factors. It can exist as either inorganic or organic compounds and often will change forms as it moves through the various media. The chemical and physical properties depend on the state of the metalloid. Only the properties of metallic arsenic have been listed; properties of other arsenic compounds are often quite different.

CAS Number: 7440-38-2

Chemical Formula: As

IUPAC Name: Arsenic

Chemical and Physical Properties

Atomic Weight: 74.91

Boiling Point: 613°C

Melting Point: 817°C

Specific Gravity: 5.72 at 20°C

67

Solubility in Water: Insoluble; some salts are soluble

Transport and Fate

In the natural environment, arsenic has four different oxidation states, and chemical speciation is important in determining arsenic's distribution and mobility. Interconversions of the +3 and +5 states as well as organic complexation, are the most important. Arsenic is generally quite mobile in the environment. In the aquatic environment, volatilization is important when biological activity or highly reducing conditions produce arsine or methylarsenics. Sorption by the sediment is an important fate for the chemical. Arsenic is metabolized to organic arsenicals by a number of organisms; this increases arsenic's mobility in the environment. Because of its general mobility, arsenic tends to cycle through the environment. Its ultimate fate is probably the deep ocean, but it may pass through numerous stages before finally reaching the sea.

Health Effects

Arsenic has been implicated in the production of skin cancer in humans. There is also extensive evidence that inhalation of arsenic compounds causes lung cancer in workers. Arsenic compounds cause chromosome damage in animals, and humans exposed to arsenic compounds have been reported to have an elevated incidence of chromosome aberrations. Arsenic compounds have been reported to be teratogenic, fetotoxic, and embryotoxic in several animal species, and an increased incidence of multiple malformations among children born to women occupationally exposed to arsenic has been reported. Arsenic compounds also cause noncancerous, possibly precancerous, skin changes in exposed individuals. Several cases of progressive polyneuropathy involving motor and sensory nerves and particularly affecting the extremities and myelinated long-axon neurons have been reported in individuals occupationally exposed to inorganic arsenic. Polyneuropathies have also been reported after the ingestion of arsenic-contaminated foods.

Toxicity to Wildlife and Domestic Animals

Various inorganic forms of arsenic appear to have similar levels of toxicity; they all seem to be much more toxic than organic forms. Acute toxicity to adult freshwater animals occurs at levels of arsenic trioxide as low as 812 µg/liter and at levels as low as 40 µg/liter in early life stages of aquatic organisms. Acute toxicity to saltwater fish occurs at levels around 15 mg/liter, while some invertebrates are affected at much lower levels (508 µg/liter). Arsenic toxicity does not appear to increase greatly with chronic exposure, and it does not seem that arsenic is bioconcentrated to a great degree.

Arsenic poisoning is a rare but not uncommon toxic syndrome among domestic animals. Arsenic causes hyperemia and edema of the gastrointestinal tract, hemorrhage of the cardiac serosal surfaces and peritoneum, and pulmonary congestion and edema; and it may cause liver necrosis. Information on arsenic toxicity to terrestrial wildlife was not reported in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 440 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 508 μg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of arsenic in water are:

Risk	Concentration
10^{-5}	22 ng/liter
10^{-6}	2.2 ng/liter
10^{-7}	0.22 ng/liter

CAG Unit Risk (USEPA): 15 $(\text{mg/kg/day})^{-1}$

National Interim Primary Drinking Water Standard (USEPA): 50 μg/liter

NIOSH Recommended Standard (air): 2 $\mu g/m^3$ Ceiling Level

OSHA Standard (air): 500 $\mu g/m^3$ TWA

ACGIH Threshold Limit Value: 200 $\mu g/m^3$ (soluble compounds, as As)

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ASBESTOS

Summary

Asbestos is a collective term applied to numerous fibrous mineral compounds of natural origin. These compounds are quite stable in the environment and may move either by wind dispersion or by being transported in surface water. Asbestos is a human carcinogen and causes lung tumors and mesotheliomas in persons exposed by inhalation. Asbestosis, a progressive, irreversible lung disease is also caused by exposure to asbestos fibers.

Background Information

Asbestos is a collective mineralogical term applied to numerous fibrous mineral silicates composed of silicon, oxygen, hydrogen, and metal cations such as sodium, magnesium, calcium, and iron. There are two major groups of asbestos: serpentine (chrysotile) and amphibole (amosite, crocidolite, anthophyllite, tremolite, and actinolite). The chemical composition of different asbestos fibers varies widely, as do the physical and chemical properties. Asbestos fibers are resistant to fire and to most solvents, but they will deteriorate rapidly in some reagents (strong acids and bases) at temperatures greater than about 95 to 100°C.

CAS Number: 1332-21-4

Transport and Fate

Asbestos is stable and is not prone to significant chemical or biological degradation in the aquatic environment. After introduction into surface waters, it remains in suspension until physical degradation or chemical coagulation allows it to settle into the sediment layer. The importance of transport from the surface of aquatic environments by wind-activated aerosol formation is not known. However, mobilization of asbestos from terrestrial surfaces and soils into the atmosphere by wind is known to occur. Of 243,527 metric tons of asbestos discharged to the environment in the United States in 1975, 98.3% was discharged to land, 1.5% to air, and 0.2% to water.

Health Effects

The severity of health effects associated with asbestos depends on several factors including fiber length and diameter, the number of fibers, fiber degradation and retention, and asbestos type. The relative pathogenicity of asbestos fibers with different characteristics appears to be variable. However, all asbestos produces adverse health effects, and therefore, it is generally considered as a single entity.

Asbestos is a recognized human carcinogen causing lung cancer and mesothelioma, a form of neoplasm of the lining of the chest and abdominal cavities, in workers exposed by inhalation. Mesotheliomas have also been identified in individuals living near asbestos plants. Excesses of cancers of the gastrointestinal tract have been identified in asbestos-exposed workers; it is unclear if exposure is via ingestion or via the passage of phagocytized particles through the body from the lung. Cancer of the larynx has also been associated with exposure to asbestos. Cigarette smoking potentiates the risk of lung cancer in individuals exposed to asbestos. All commercial forms of asbestos are carcinogenic in experimental animals.

No data exist on the teratogenicity of asbestos, although transplacental transfer of asbestos has been reported. In a study using several chrysotile and crocidolite samples, both transformation of morphology and positive genetic responses resulted from passive inclusion of asbestos in the culture media of Chinese hamster cells, although very fine fibrous glass produced the same abnormalities. No mutagenicity has been observed in any bacterial test systems.

Long-term exposure to asbestos dust also causes asbestosis, a progressive, irreversible lung disease characterized by diffuse interstitial fibrosis. Acute effects are of little consequence after exposure to high concentrations via inhalation although some temporary breathing difficulty has occasionally been reported by workers. Although human data on initial changes are unavailable, local inflammatory lesions are found in the terminal bronchioles of rats following inhalation. Progressive fibrosis follows within a few weeks of the direct exposure to dust. In experiments with rats, cellular proliferation and DNA synthesis in the stomach, duodenum, and jejunum appear to be an immediate response to asbestos ingestion. Structural changes in the ileum, particularly the villi, are also observed.

Toxicity to Wildlife and Domestic Animals

No data concerning the effects of asbestos on wildlife and domestic animals are available. Tissue samples of freshwater fish species from water with known asbestos contamination contain asbestos fibers identical to those in the water. Muscle tissue concentrations were about one-twelfth of the average water concentrations, but liver and kidney fiber concentrations were 500 times greater than muscle tissue concentrations.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of carcinogenic risks associated with lifetime exposure to various concentrations of asbestos in water are:

Risk	Concentration
10^{-5}	300,000 fibers/liter
10^{-6}	30,000 fibers/liter
10^{-7}	3,000 fibers/liter

NIOSH Recommended Standards:

0.1 fibers/ml as an 8-hr TWA
0.5 fibers/ml as a 15-min Ceiling Level

OSHA Standards:

2 fibers longer than 5 μm in length per ml of air as an 8-hour TWA
10 fibers/ml as a 10-min Ceiling Level

ACGIH Threshold Limit Values:

Amosite, 0.5 fibers greater than 5 μm in length/ml

Chrysotile, 2 fibers greater than 5 μm in length/ml

Crocidolite, 0.2 fibers greater than 5 μm in length/ml

Other forms, 2 fibers greater than 5 μm in length/ml

Asbestos is considered a recognized human carcinogen.

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BARIUM

Summary

In its pure form, barium is an extremely reactive metal that decomposes in water. In natural waters it forms insoluble carbonate or sulfate salts and is usually present at concentrations of less than 1 mg/liter. Insoluble forms of barium are not very toxic; but soluble barium salts are highly toxic after acute exposure, and they have a prolonged stimulant effect on muscles. A benign pneumoconiosis, baritosis, can result from inhaling barium dusts. The EPA Interim Primary Drinking Water Standard is 1 mg/liter.

CAS Number: 7440-39-3

Chemical Formula: Ba

IUPAC Name: Barium

Chemical and Physical Properties

Atomic Weight: 137.3

Boiling Point: 1,640°C

Melting Point: 725°C

Specific Gravity: 3.5

Solubility in Water: Decomposes; combines with sulfate present in natural waters to form $BaSO_4$, which has a solubility of 1.6 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol; insoluble in benzene

Transport and Fate

Barium is extremely reactive, decomposes in water, and readily forms insoluble carbonate and sulfate salts. Barium is generally present in solution in surface or groundwater only in trace amounts. Large amounts will not dissolve because natural waters usually contain sulfate, and the solubility of barium sulfate is generally low. Barium is not soluble at more than a few parts per million in water that contains sulfate at more than a few parts per million. However, barium sulfate may become considerably more soluble in the presence of chloride and other anions. Monitoring programs show that it is rare to find barium in drinking water at concentrations greater than 1 mg/liter. Atmospheric transport of barium, in the form of particulates, can occur. Bioaccumulation is not an important process for barium.

Health Effects

There are no reports of carcinogenicity, mutagenicity, or teratogenicity associated with exposure to barium or its compounds. Effects on gametogenesis and on the reproductive organs are reported in male and female rats after inhalation of barium carbonate; intratesticular injection of barium chloride affects the male reproductive organs.

Insoluble forms of barium, particularly barium sulfate, are not toxic by ingestion or inhalation because only minimal amounts are absorbed. However, soluble barium compounds are highly toxic in humans after exposure by either route. The most important effect of acute barium poisoning is a strong, prolonged stimulant action on muscle. Smooth, cardiac, and skeletal muscles are all affected, and a transient increase in blood pressure due to vasoconstriction can occur. Effects on the hematopoietic system and cerebral cortex have also been reported in humans. Accidental ingestion of soluble barium salts has resulted in gastroenteritis, muscular paralysis, and ventricular fibrillation and extra systoles. Potassium deficiency can occur in cases of acute poisoning. Doses of barium carbonate and barium chloride of 57 mg/kg and 11.4 mg/kg, respectively, have been reported to be fatal in humans. Digitalis-like toxicity, muscle stimulation, and effects on the hematopoietic and central nervous systems have been confirmed in experimental animals. There are no adequate animal data available for determining the chronic effects of low level exposure to barium by ingestion.

Baritosis, a benign pneumoconiosis, is an occupational disease arising from the inhalation of barium sulfate dust, barium oxide dust, and barium carbonate. The radiologic changes produced in the lungs are reversible with cessation of exposure. Other reports of industrial exposure to barium compounds describe pulmonary nodulation with or without a decrease in lung function. Dusts of barium oxide are considered potential agents of dermal and nasal irritation. The biological half-life for barium is less than 24 hours.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity to wildlife and domestic animals are not available.

Regulations and Standards

Interim Primary Drinking Water Standard: 1 mg/liter

OSHA Standard: 0.5 mg/m^3 (soluble compounds, as Ba)

ACGIH Threshold Limit Value: 0.5 mg/m^3 (soluble compounds, as Ba)

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BENZENE

Summary

Benzene is an important industrial solvent and chemical intermediate. It is rather volatile, and atmospheric photooxidation is probably an important fate process. Benzene is a known human carcinogen, causing leukemia in exposed individuals. It also adversely affects the hematopoietic system. Benzene has been shown to be fetotoxic and to cause embryolethality in experimental animals. Exposure to high concentrations of benzene in the air causes central nervous system depression and cardiovascular effects, and dermal exposure may cause dermatitis.

CAS Number: 71-43-2

IUPAC Name: Benzene

Chemical Formula: C_6H_6

Chemical and Physical Properties

Molecular Weight: 78.12

Boiling Point: 80.1°C

Melting Point: 5.56°C

Specific Gravity: 0.879 at 20°C

Solubility in Water: 1,780 mg/liter at 25°C

Solubility in Organics: Miscible with ethanol, ether, acetic acid, acetone, chloroform, carbon disulfide, and carbon tetrachloride

Log Octanol/Water Partition Coefficient: 1.95-2.13

Vapor Pressure: 75 mm Hg at 20°C

Vapor Density: 2.77

Flash Point: -11.1°C

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Transport and Fate

Volatilization appears to be the major transport process of benzene from surface waters to the ambient air, and atmospheric transport of benzene occurs readily (USEPA 1979). Although direct oxidation of benzene in environmental waters is unlikely, cloud chamber data indicate that it may be photooxidized rapidly in the atmosphere. Inasmuch as volatilization is likely to be the main transport process accounting for the removal of benzene from water, the atmospheric destruction of benzene is probably the most likely fate process. Values for benzene's log octanol/water partition coefficient indicate that adsorption onto organic material may be significant under conditions of constant exposure. Sorption processes are likely removal mechanisms in both surface water and groundwater. Although the bioaccumulation potential for benzene appears to be low, gradual biodegradation by a variety of microorganisms probably occurs. The rate of benzene biodegradation may be enhanced by the presence of other hydrocarbons.

Health Effects

Benzene is a recognized human carcinogen (IARC 1982). Several epidemiological studies provide sufficient evidence of a causal relationship between benzene exposure and leukemia in humans. Benzene is a known inducer of aplastic anemia in humans, with a latent period of up to 10 years. It produces leukopenia and thrombocytopenia, which may progress to pancytopenia. Similar adverse effects on the blood-cell-producing system occur in animals exposed to benzene. In both humans and animals, benzene exposure is associated with chromosomal damage, although it is not mutagenic in microorganisms. Benzene was fetotoxic and caused embryolethality in experimental animals.

Exposure to very high concentrations of benzene [about 20,000 ppm (66,000 mg/m^3) in air] can be fatal within minutes (IARC 1982). The prominent signs are central nervous system depression and convulsions, with death usually following as a consequence of cardiovascular collapse. Milder exposures can produce vertigo, drowsiness, headache, nausea, and eventually unconsciousness if exposure continues. Deaths from cardiac sensitization and cardiac arrhythmias have also been reported after exposure to unknown concentrations. Although most benzene hazards are associated with inhalation exposure, dermal absorption of liquid benzene may occur, and prolonged or repeated skin contact may produce blistering, erythema, and a dry, scaly dermatitis.

Toxicity to Wildlife and Domestic Animals

The EC_{50} values for benzene in a variety of invertebrate and vertebrate freshwater aquatic species range from 5,300 µg/liter to 386,000 µg/liter (USEPA 1980). However, only values for the rainbow trout (5,300 µg/liter) were obtained from a flow through test and were based on measured concentrations. Results based on unmeasured concentrations in static tests are likely to underestimate toxicity for relatively volatile compounds like benzene. A chronic test with Daphnia magna was incomplete, with no adverse effects observed at test concentrations as high as 98,000 µg/liter.

For saltwater species, acute values for one fish and five invertebrate species range from 10,900 µg/liter to 924,000 µg/liter. Freshwater and saltwater plant species that have been studied exhibit toxic effects at benzene concentrations ranging from 20,000 µg/liter to 525,000 µg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest concentrations of benzene known to cause toxic effects in aquatic organisms.

Freshwater

Acute toxicity: 5,300 µg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 5,100 µg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of benzene in water are:

Risk	Concentration
10^{-5}	6.6 µg/liter
10^{-6}	0.66 µg/liter
10^{-7}	0.066 µg/liter

CAG Unit Risk (USEPA): 2.9×10^{-2} $(\mathrm{mg/kg/day})^{-1}$

OSHA Standards: 30 $\mathrm{mg/m^3}$ TWA
75 $\mathrm{mg/m^3}$ Ceiling Level
150 $\mathrm{mg/m^3}$ 10-min Peak Level

ACGIH Threshold Limit Values: Suspected human carcinogen
30 $\mathrm{mg/m^3}$ TWA
75 $\mathrm{mg/m^3}$ STEL

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BENZIDINE

Summary

Benzidine is an aromatic amine that can be formed in the environment by the degradation of benzidine-based dyes. It is rapidly oxidized by metal cations in natural waters to form radical cations which may be fairly persistent. Benzidine is considered to be a human carcinogen; epidemiological studies show that it causes bladder cancer. It has also caused liver and bladder tumors in animals and it is mutagenic in bacterial test systems. Exposure to benzidine causes noncarcinogenic liver and kidney damage in animals exposed by various routes. Benzidine is toxic to aquatic life at concentrations as low as 2,500 µg/liter.

CAS Number: 92-87-5

Chemical Formula: $C_{12}H_8(NH_2)_2$

IUPAC Name: Benzidine

Important Synonyms and Trade Names: p-Benzidine; 4,4'-Diaminobiphenyl

Chemical and Physical Properties

Molecular Weight: 184.23

Boiling Point: 402°C

Melting Point: 129°C

Specific Gravity: 1.250

Solubility in Water: 400 mg/liter at 12°C

Solubility in Organics: Soluble in alcohol and ether

Log Octanol/Water Partition Coefficient: 1.81

Vapor Density: 6.36

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Transport and Fate

The physical and chemical properties of benzidine suggest that direct photolysis of this compound probably occurs in aquatic systems. Photooxidation and direct interaction with molecular oxygen or hydroperoxy radicals probably are significant fates for benzidine. However, oxidation by the metal cations of natural waters, such as Fe III, Al III, and Cu II, is probably the most rapid fate process in aquatic systems. Hydrolysis and volatilization do not appear to be important environmental fate processes. Although the moderate log octanol/water partition coefficient of benzidine suggests little potential for sorption by organic particulates, adsorption to clay minerals and metal cation complexes is very rapid and may be the most important environmental transport process for this compound. It is suggested that intercalation of a benzidine radical-cation into clay particles may increase its stability in environmental waters. These particles could then become part of the bed sediment or could be transported in surface water systems without being detected by most analytical methods for measuring benzidine levels. Bioaccumulation does not appear to be an important environmental process for benzidine. Benzidine is metabolized by mammals, and detoxification probably proceeds by acetylation of the amino groups. Biodegradation by activated sludge may contribute slightly to the degradation of benzidine during sewage plant treatment. The extent of microbial degradation of benzidine in natural waters is unknown.

Health Effects

Benzidine is considered a human carcinogen. Epidemiological studies show a strong association between occupational exposure to benzidine and development of bladder cancer. Exposure can occur through ingestion, inhalation, or percutaneous absorption. Benzidine is also carcinogenic in experimental animals. For example, oral exposure produces liver tumors in the rat and hamster, and bladder tumors in the dog. Benzidine has been shown to be mutagenic in a variety of test systems. No reports on its teratogenicity or reproductive effects are available.

Relatively litte information is available concerning the noncarcinogenic effects of benzidine because most experimental work is concerned with evaluating the carcinogenic risk of the compound. Nausea, vomiting, and possibly liver and kidney damage are reported to be possible effects due to ingestion. In experimental animals, liver and kidney damage are often observed after chronic or subchronic exposure by various routes.

Toxicity to Wildlife and Domestic Animals

Available data indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 2,500 μg/liter and would occur at lower concentrations among species more sensitive than those tested.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of benzidine in water are:

Risk	Concentration
10^{-5}	1.2 ng/liter
10^{-6}	0.12 ng/liter
10^{-7}	0.01 ng/liter

CAG Unit Risk (USEPA): 234 $(mg/kg/day)^{-1}$

NIOSH Recommended Standard: Human carcinogen

OSHA Standard: Human carcinogen

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BENZO(a)ANTHRACENE

Summary

Benzo(a)anthracene is a four-ringed polycylic aromatic hydrocarbon (PAH). It is readily absorbed to organic matter and is probably moderately persistent in the environment. Benzo(a) anthracene is carcinogenic in mice and is reported to be mutagenic in several test systems. Carcinogenic PAHs such as benzo(a) anthracene cause immunosuppression, and dermal exposure causes chronic dermatitis and other skin disorders. The very limited information on its toxicity to aquatic life indicates that benzo(a)anthracene is chronically toxic to fish at concentrations of less than 1,000 µg/liter.

CAS Number: 56-55-3

Chemical Formula: $C_{18}H_{12}$

IUPAC Name: 1,2-benzanthracene

Important Synonyms and Trade Names: 1,2-Benzanthracene; 2,3-Benzophenanthrene; Benzo(b)phenanthrene

Chemical and Physical Properties

Molecular Weight: 228.28

Melting Point: 155-157°C

Solubility in Water: 0.009 to 0.014 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, ether, acetone, and benzene

Log Octanol/Water Partition Coefficient: 5.61

Vapor Pressure: 5×10^{-9} mm Hg at 20°C

Transport and Fate

Dissolved benzo(a)anthracene can undergo rapid, direct photolysis, and this process may be an important environmental fate in aquatic systems. Studies indicate that singlet oxygen is the oxidant and that quinones are the products in the photolytic reactions. The free-radical oxidation of benzo(a)anthracene in the environment is rapid and may be competitive with photolysis as a chemical fate process. When chlorine and ozone are present in aquatic systems in sufficient quantities, oxidation reactions resulting in the formation of quinones may be significant fate processes. Because benzo(a)anthracene does not contain groups amenable to hydrolysis, this process is not thought to be a significant environmental fate. Volatilization does not appear to be an important transport process either.

Available information indicates that benzo(a)anthracene will accumulate in the sediment and biotic portions of the aquatic environment and that adsorption to suspended matter is the dominant transport process. Sorption onto sediments, soil particles, and biota is strongly correlated with the organic carbon levels present. Although benzo(a)anthracene is readily and rapidly bioaccumulated, it is also rapidly metabolized and excreted. Therefore, bioaccumulation is short term and is not considered an important fate process. Benzo(a)anthracene is degraded by microbes and readily metabolized by multicellular organisms. Degradation by mammals is considered to be incomplete; the parent compound and metabolites are excreted by the urinary system. Biodegradation is probably the ultimate fate process for benzo(a)anthracene. It generally is more rapid in soil than in aquatic systems and is relatively fast in those systems chronically affected by polycyclic aromatic hydrocarbon contamination.

Atmospheric transport of benzo(a)anthracene can occur, and the chemical can be returned to aquatic and terrestrial systems by atmospheric fallout or with precipitation. Benzo(a)anthracene can also enter surface and groundwater by leaching from polluted soils.

Health Effects

Benzo(a)anthracene administered by different routes is carcinogenic in the mouse. It can produce hepatomas and lung adenomas following repeated oral administration and bladder tumors following implantation. Benzo(a)anthracene can also produce tumors in mice following subcutaneous injections. Although benzo(a)anthracene is a complete carcinogen for mouse skin, it produces less skin tumors with a longer latency than does benzo(a)pyrene. Benzo(a)anthracene has not been adequately tested in other species.

Benzo(a)anthracene is reported to be mutagenic in a variety of test systems. In some cases, a correlation is observed between mutagenicity and carcinogenic potency for benzo(a)anthracene and other polycyclic aromatic hydrocarbons. In other words, those compounds exhibiting greater mutagenic activity often have higher carcinogenic potency as well. No adequate information concerning the teratogenic effects of benzo(a)anthracene in humans or experimental animals is available.

Application of the carcinogenic polycyclic aromatic hydrocarbons, including benzo(a)anthracene, to mouse skin leads to the destruction of sebaceous glands, hyperplasia, hyperkeratosis, and ulceration. Workers exposed to materials containing polynuclear aromatic hydrocarbons may exhibit chronic dermatitis, hyperkeratoses, and other skin disorders. Repeated subcutaneous injections of benzo(a)anthracene to mice and rats produces gross changes in the lymphoid tissues. It has also been shown that many carcinogenic polycyclic aromatic hydrocarbons can produce an immunosuppressive effect, although specific results with benzo(a)anthracene have not been reported.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity to wildlife and domestic animals are not available. One study involving freshwater fish reported an 87% mortality rate in bluegills exposed to 1,000 μg/liter benzo(a)anthracene for 6 months.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

Risk	Concentration
10^{-5}	28 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

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BENZOTHIAZOLE

Summary

Benzothiazole has a moderate acute toxicity.

CAS Number: 95-16-9

Chemical Formula: C_6H_4SCHN

IUPAC Name: 2-Benzothiazole

Important Synonyms and Trade Names: Benzosulfonaxole, 0-2857, 1-Thia-3-azaindene, USAF EK-4812, and 2-benzothiazole

Chemical and Physical Properties

Molecular Weight: 135.19

Boiling Point: 227°C

Specific Gravity: 1.246 at 20°C

Solubility in Water: Slightly soluble in water

Solubility in Organics: Freely soluble in alcohol and carbon disulfide

Log Octanol/Water Partition Coefficient: 2.01

Transport and Fate

No information on the transport and fate of benzothiazole was available in the sources reviewed.

Health Effects

Very few data on the toxicity of benzothiazole were found in the literature searched. The oral LD_{50} in the mouse is 900 mg/kg, and the intravenous injection LD_{50} value is 95 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of benzothiazole to wildlife and domestic animals was found in the sources reviewed.

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BERYLLIUM

Summary

Beryllium is a metal with a complicated coordination chemistry, and it can form complexes, oxycarboxylates, and chelates with a variety of materials. Inhalation exposure to beryllium causes lung and bone cancer in animals, and epidemiological studies suggest that it may cause lung cancer in humans. Acute respiratory effects are associated with inhalation of beryllium, and dermal exposure can cause contact dermatitis. Chronic exposure to beryllium was reported to have adverse effects on aquatic organisms at levels as low as 5.3 μg/liter.

CAS Number: 7440-41-7

Chemical Formula: Be

IUPAC Name: Beryllium

Chemical and Physical Properties (Metal)

Atomic Weight: 9.012

Boiling Point: 2970°C

Melting Point: 1278°C

Specific Gravity: 1.85 at 20°C

Solubility in Water: Insoluble; most salts are soluble

Solubility in Organics: Soluble in dilute acid and alkali; insoluble in alcohol, ether, and CCl_4

Transport and Fate

Most common beryllium compounds are readily soluble in water. However, in water, soluble beryllium salts are hydrolyzed to form beryllium hydroxide. The solubility of beryllium hydroxide is quite low (2 mg/liter) in the pH range of most natural waters. Formation of hydrated complexes may increase the solubility of beryllium somewhat, especially at higher pH where polynuclear hydroxide complexes may form. It is probable, however, that in most natural aquatic environments beryllium is present in particulate rather than dissolved form.

Although little information concerning adsorption of beryllium is available, based on its geochemical similarity to aluminum it is expected to be adsorbed onto clay mineral surfaces at low pH and to be complexed into some insoluble compounds at high pH. In most natural environments, beryllium is likely to be present in sorbed or precipitated, rather than dissolved, form.

Beryllium may be accumulated to a slight extent by aquatic organisms. Although it has a low solubility in water, it is possible that benthos could accumulate beryllium from sediment and thereby transfer the metal to higher organisms via the food chain. However, there is no evidence for food chain magnification. Airborne transport of beryllium, generally in the form of particulates, may also occur.

Health Effects

The results of some epidemiological studies of workers occupationally exposed to beryllium indicate that beryllium may cause lung cancer in humans. Although this evidence is equivocal, beryllium and many of its compounds are known to be carcinogenic in several animal species. Inhalation exposure to beryllium has resulted in the development of lung or bone cancer in animals, and exposure by injection has produced bone cancer. Although beryllium compounds may impair DNA polymerization, there is no other evidence of mutagenic or clastogenic activity. However, the number of compounds tested and the types of tests conducted have been limited. There is little information concerning the possible teratogenic effects of beryllium. It is reported to inhibit embryonic development of the snail and regeneration of the limbs of the salamander.

Acute respiratory effects due to beryllium exposure include rhinitis, pharyngitis, tracheobronchitis, and acute pneumonitis. Dermal exposure to soluble beryllium compounds can cause contact dermatitis. Ocular effects include inflammation of the conjunctiva from splash burns or in association with contact dermatitis. The most common clinical symptoms caused by chronic beryllium exposure are granulomatous lung inflammation, with accompanying cough, chest pain, and general weakness. Systemic effects include right heart enlargement with accompanying cardiac failure, liver and spleen enlargement, cyanosis, digital clubbing, and kidney stone development.

Toxicity to Wildlife and Domestic Animals

Data for several freshwater fish species indicate that the acute toxicity of beryllium decreases by about two orders of magnitude with an increase in hardness from about 20 to

400 mg/liter calcium carbonate. For example, acute values for the fathead minnow range from 150 to 20,000 µg/liter over this range of hardness. There does not appear to be much variation in sensitivity among the fish species tested at similar levels of hardness. Acute and chronic values for the invertebrate Daphnia magna in the same test water (hardness equal to 220 mg/liter) were reported to be 2,500 and 5.3 µg/liter, respectively, indicating a very large difference between acute and chronic toxicity. Only limited, inconclusive data exist concerning beryllium toxicity in saltwater species. Growth of the green alga Chlorella vannieli is inhibited at a beryllium concentration of 100,000 µg/liter. A bioconcentration factor of 19 with a half-life of one day in the whole body is reported for the bluegill.

Some toxicity due to beryllium has been seen in domestic animals. One of the earliest observed effects of beryllium toxicity was the development of rachitic bone changes after the addition of soluble beryllium salts to the diet of poultry and livestock. Approximately 0.125% beryllium carbonate in the food or water is required to produce a mild case.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest concentrations of beryllium known to cause toxic effects in aquatic organisms.

Freshwater

Acute toxicity: 130 µg/liter
Chronic toxicity: 5.3 µg/liter

Saltwater

Acute toxicity: No available data
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of beryllium in water are:

Risk	Concentration
10^{-5}	37 ng/liter
10^{-6}	3.7 ng/liter
10^{-7}	0.37 ng/liter

CAG Unit Risk (USEPA): 2.6 $(mg/kg/day)^{-1}$

OSHA Standards (air): 2 $\mu g/m^3$ TWA
5 $\mu g/m^3$ Ceiling Level
25 $\mu g/m^3$/30 min Peak Concentration

ACGIH Threshold Limit Value: Suspected human carcinogen
2 $\mu g/m^3$

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1-BUTANOL

Summary

1-Butanol, or n-butyl alcohol, is a short-chain alcohol. It is very soluble in water and is likely to be moderately persistent in the environment. Butanol is irritating to the eyes and mucous membranes. It is not very toxic to aquatic life; the lowest dose reported to adversely affect fish was 1,000 mg/liter in the creek chub.

CAS Number: 71-36-3

Chemical Formula: C_4H_9OH

IUPAC Name: 1-Butanol

Important Synonyms and Trade Names: n-Butanol, n-butyl alcohol, propyl carbinol

Chemical and Physical Properties

Molecular Weight: 74.12

Boiling Point: 117.7°C

Melting Point: -89.9°C

Specific Gravity: 0.810 at 20°C

Solubility in Water: 77,000 mg/liter at 25°C

Solubility in Organics: Miscible with alcohol, ether, and many other organic solvents

Log Octanol/Water Partition Coefficient: 1.0 (calculated)

Vapor Pressure: 4.4 mm Hg at 20°C

Vapor Density: 2.55

Flash Point: 36-38°C

Transport and Fate

No information on the transport and fate of butanol was found in the sources reviewed. However, based on the general reactions of alcohols and the specific chemical and physical properties of the material, likely transport and fate process can be determined.

Alcohols are very soluble in water and therefore probably are not very volatile, although some evaporation may occur. Oxidation is likely to be an important fate process in both surface water and the atmosphere. In soil, butanol is probably biodegraded by soil microorganisms.

Health Effects

The information on the health effects of butanol is limited. No data on the carcinogenicity, mutagenicity, or reproductive toxicity of butanol were found in the literature reviewed. Workers exposed to butyl alcohol had greater hearing loss than unexposed individuals. After several hours of exposure, 600 mg/kg of butanol irritates the eyes and mildly irritates the mucous membranes in humans. The oral LD_{50} in rats was reported to be 790 mg/kg.

Toxicity to Wildlife and Domestic Animals

The 24-hour LD_0 and LD_{100} values for butanol in the creek chub were 1,000 and 1,400 mg/liter, respectively. Butanol was toxic to the alga Ehlorella pyrenoidosa at 8,500 mg/liter.

No information on the toxicity of butanol to terrestrial wildlife or domestic animals was found in the sources examined.

Regulations and Standards

OSHA Standard (air): 300 mg/m^3 TWA

ACGIH Threshold Limit Value: 150 mg/m^3 Ceiling level

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CADMIUM

Summary

Cadmium is a metal that can be present in a variety of chemical forms in wastes or in the environment. Some forms are insoluble in water, but cadmium is relatively mobile in the aquatic environment. Cadmium is carcinogenic in animals exposed by inhalation and may also be in humans. It is uncertain whether it is carcinogenic in animals or humans exposed via ingestion. Cadmium is a known animal teratogen and reproductive toxin. It has chronic effects on the kidney, and background levels of human exposure are thought to provide only a relatively small margin of safety for these effects.

Background Information

Cadmium is a soft, bluish white metal that is obtained as a by-product from the treatment of the ores of copper, lead, and iron. Cadmium has a valence of +2 and has properties similar to those of zinc. Cadmium forms both organic and inorganic compounds. Cadmium sulfate is the most common salt.

CAS Number: 7440-43-9

Chemical Formula: Cd

IUPAC Name: Cadmium

Chemical and Physical Properties

Atomic Weight: 112.41

Boiling Point: 765°C

Melting Point: 321°C

Specific Gravity: 8.642

Solubility in Water: Salts are water soluble; metal is insoluble

Solubility in Organics: Variable, based on compound

Vapor Pressure: 1 mm Hg at 394°C

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Transport and Fate

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals (USEPA 1979). It is removed from aqueous media by complexing with organic materials and subsequently being adsorbed to the sediment. It appears that cadmium moves slowly through soil, but only limited information on soil transport is available. Cadmium uptake by plants is not a significant mechanism for depletion of soil accumulations but may be significant for human exposure.

Health Effects

There is suggestive evidence linking cadmium with cancer of the prostate in humans (USEPA 1980). In animal studies, exposure to cadmium by inhalation caused lung tumors in rats, and exposure by injection produced injection-site sarcomas and/or Leydig-cell tumors (Takenaka 1983, USEPA 1981). An increased incidence of tumors has not been seen in animals exposed to cadmium orally, but four of the five available studies were inadequate by current standards (Clement 1983).

The evidence from a large number of studies on the mutagenicity of cadmium is equivocal, and it has been hypothesized that cadmium is not directly mutagenic but impedes repair (Clement 1983). Cadmium is a known animal teratogen and reproductive toxin. It has been shown to cause renal dysfunction in both humans and animals. Other toxic effects attributed to cadmium include immunosuppression (in animals), anemia (in humans), pulmonary disease (in humans), possible effects on the endocrine system, defects in sensory function, and bone damage. The oral LD_{50} in the rat was 225 mg/kg (NIOSH 1983).

Toxicity to Wildlife and Domestic Animals

Laboratory experiments suggest that cadmium may have adverse effects on reproduction in fish at levels present in lightly to moderately polluted waters.

The acute LC_{50} for freshwater fish and invertebrates generally ranged from 100 to 1,000 μg/liter; salmonids are much more sensitive than other organisms (USEPA 1980). Saltwater species were in general 10-fold more tolerant to the acute effects of cadmium. Chronic tests have been performed and show that cadmium has cumulative toxicity and acute-chronic ratios that range of from 66 to 431. Bioconcentration factors were generally less than 1,000 but were as high as 10,000 for some freshwater fish species.

No adverse effects on domestic or wild animals were reported in the studies reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed 1984)

Freshwater

Acute toxicity: $e^{(1.30[\ln(\text{hardness})] - 3.92)}$ µg/liter

Chronic toxicity: $e^{(0.87[\ln(\text{hardness})] - 4.38)}$ µg/liter

Saltwater

Acute toxicity: 38 µg/liter
Chronic toxicity: 12 µg/liter

Human Health

Criterion: 10 µg/liter

CAG Unit Risk for inhalation exposure (USEPA): 6.1 $(\text{mg/kg/day})^{-1}$

Interim Primary Drinking Water Standard (USEPA): 10 µg/liter

NIOSH Recommended Standards: 40 $\mu g/m^3$ TWA
200 $\mu g/m^3$/15 min Ceiling Level

OSHA Standards: 200 $\mu g/m^3$ TWA
600 $\mu g/m^3$ Ceiling Level

ACGIH Threshold Limit Values: 50 $\mu g/m^3$ TWA

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CARBON TETRACHLORIDE

Summary

Carbon tetrachloride is used as an industrial solvent and chemical intermediate. It is an animal carcinogen, causing liver tumors in mice, rats, and hamsters. Carbon tetrachloride also causes liver and kidney damage in animals and humans.

Chemical Formula: CCl_4

IUPAC Name: Tetrachloromethane

Important Synonyms and Trade Names: Tetrachloromethane, perchloromethane

Chemical and Physical Properties

Molecular Weight: 153.8

Boiling Point: 76.7°C

Melting Point: 22.9°C

Specific Gravity: 1.59 at 20°C (liquid)
5.3 vapor (gas) specific gravity

Solubility in Water: 800 mg/liter

Solubility in Organics: Miscible with alcohol, benzene, chloroform, ether, and carbon disulfide

Log Octanol/Water Partition Coefficient: 2.64

Vapor Pressure: 90 mm Hg at 20°C

Vapor Density: 5.32

Transport and Fate

Carbon tetrachloride has a high vapor pressure and therefore volatilizes rapidly into the atmosphere from surface water and probably from soil. It is relatively soluble in water and therefore would be expected to be transported in groundwater. Because of its high specific gravity, it may move independently from the groundwater as a nonaqueous phase liquid.

Health Effects

Carbon tetrachloride was carcinogenic in mice, rats, and hamsters; in all cases liver tumors were induced (IARC 1979, USEPA 1980). In addition, mice also displayed a high incidence of tumors of the adrenal gland (Weisburger 1977). Studies discussed by EPA (1980) on the mutagenic and teratogenic effects of carbon tetrachloride and its impact on reproduction are inconclusive. Carbon tetrachloride also causes both liver and kidney damage in animals and humans. One study in which guinea pigs were repeatedly exposed to carbon tetrachloride vapor for several months provided evidence of damage to the optic nerve and degeneration of the myelin sheath of the sciatic nerve (Smyth et al. 1936).

Toxicity to Wildlife and Domestic Animals

Carbon tetrachloride has been shown to be acutely toxic to aquatic species at concentrations as low as 35 mg/liter. No data on chronic toxicity to aquatic life were reported in the literature reviewed. Fish bioconcentrate carbon tetrachloride by a factor of less than 50. No studies on the toxicity of carbon tetrachloride to domestic animals or terrestrial wildlife were found in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 35,200 μg/liter
Chronic toxicity: No available data

Saltwater:

Acute toxicity: 50,000 μg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to carbon tetrachloride at various concentrations in water are:

Risk	Concentration
10^{-5}	4.0 μg/liter
10^{-6}	0.4 μg/liter
10^{-7}	0.04 μg/liter

CAG Unit Risk (USEPA): 1.3×10^{-1} $(mg/kg/day)^{-1}$

OSHA Standards (air): 10 ppm TWA
25 ppm Ceiling Level

REFERENCES

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CHLORDANE

Summary

Chlordane is an organochlorine pesticide that was formerly used on field crops and is presently used to control structural pests in homes. Technical chlordane is a complex mixture that includes two isomers of chlordane, heptachlor, and two isomers of nonachlor. It is very persistent in the environment and is strongly bioaccumulated in fish and other aquatic organisms. Chlordane causes liver tumors in mice, and the results of a mutagenicity assay were positive. It also has adverse reproductive effects in mice, and chronic exposure causes liver changes and adversely affects the central nervous system. Chlordane is very toxic to aquatic organisms.

Background Information

Technical chlordane is a complex mixture, the major components of which are cis-chlordane and trans-chlordane. The technical product also contains a variety of other chlorinated hydrocarbons, including heptachlor. It is a viscous amber-colored liquid. Much of the available literature does not distinguish between the chlordane isomers and appears to discuss mixtures of these compounds.

CAS Number: Chlordane (mixture): 57-74-9
cis-Chlordane: 5103-74-2
trans-Chlordane: 5103-71-9

Chemical Formula: $C_{10}H_6Cl_8$

IUPAC Name: 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene

Important Synonyms and Trade Names: cis-Chlordane: alpha-Chlordane
trans-Chlordane: gamma-Chlordane

Chemical and Physical Properties

Molecular Weight: 409.8

Boiling Point: 175°C at 2 mm Hg

Melting Point: cis-Chlordane: 107-109°C,
trans-Chlordane: 103-105°C

Specific Gravity: 1.59-1.635 at 16°C (technical chlordane)

Solubility in Water: From 0.056 to 1.85 mg/liter at 25°C

Solubility in Organics: Miscible in aliphatic and aromatic solvents (technical chlordane)

Log Octanol/Water Partition Coefficient: 2.78

Vapor Pressure: 1×10^{-5} mm Hg at 20°C (refined product)

Flash Point: Minimum 81°C (technical chlordane)

Transport and Fate

Chlordane is very persistent in the environment, resisting chemical and biological degradation into harmless substances. Chlordane in clear water is somewhat volatile, and this may be an important loss process. Less loss of chlordane from aquatic systems occurs when organics are present, and residue concentrations in sediment are often much higher than in water. Therefore, sorption to sediments is probably important in removing the chemical from the aquatic environment. Chlordane binds tightly to soil particles and persists for years in soil after surface application. However, chlordane applied as an emulsifiable concentrate is more readily volatilized than when it is applied as a granular formulation. Certain food and feed crops accumulate residues by absorption from the soil. Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur.

Health Effects

Mixtures of cis-chlordane and trans-chlordane produce liver cancer in mice. Chlordane also has mutagenic effects in at least one test system. Reproductive effects, including developmental defects and neonatal metabolic and biochemical disorders, are observed in the offspring of mice exposed to chlordane. Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects. Either isomer alone, or a mixture of the two, appears to exhibit approximately equal toxicity. Acute effects include anorexia, weight loss, tremors, convulsions, and death. Chronic exposure to chlordane causes liver changes and induces or suppresses a variety of enzyme systems. In addition, chlordane may act as a cumulative neurotoxin. The oral LD$_{50}$ in the rat is 283 mg/kg. Oxychlordane, an epoxide metabolite formed from either chlordane isomer, is significantly more acutely toxic than chlordane. The oral LD$_{50}$ of oxychlordane administered to rats in corn oil is 19 mg/kg, and it is 43 mg/kg when administered in an aqueous suspension.

Acute oral or skin exposure to chlordane can cause vomiting, seizures, electroencephalographic dysrhythmia, convulsions, and death in humans. However, most reports of human toxicity are inconclusive. Oxychlordane has been found in a high percentage of human adipose tissue samples and also in human milk samples.

Toxicity to Wildlife and Domestic Animals

The toxic effects of chlordane are seen at relatively low concentrations in some fish and invertebrate species. Chlordane also shows strong tendencies for bioaccumulation in some aquatic and terrestrial organisms. It can concentrate at levels thousands of times greater than the surrounding water medium in a variety of aquatic organisms, including bacteria, algae, daphnids, and fish. The EPA criteria for acute exposure to freshwater species is 2.4 µg/liter, and it is 0.17 µg/liter for chronic exposure. The corresponding Acute and Chronic Values for saltwater species are 0.09 µg/liter, 0.0064 µg/liter, and 0.0040 µg/liter. The Final Acute-Chronic Ratio is 14. Very little information exists concerning the biotransformation of chlordane. Although biotransformations may be important for the ultimate degradation of chlordane, these processes are likely to be very slow.

Chlordane or oxychlordane residues have been found in a wide variety of wildlife and domestic animal species, but usually at relatively low levels. Chlordane does not appear to be extensively concentrated in the higher members of the terrestrial food chain. Studies indicate that chlordane may produce toxic effects in certain soil invertebrates after surface application. Although little information concerning bioaccumulation in these organisms is available, the potential bioconcentration of chlordane or oxychlordane by terrestrial insectivores is of concern. Little information on the toxic effects of chlordane to mammalian wildlife and domestic animal species is available. Chlordane or oxychlordane residues have been found in crops, meat, fish and poultry, dairy products, and eggs. Oral LD$_{50}$ values for chlordane ranging from 331 to 858 ppm in the diet (approximately 25-50 mg/kg) are reported for a variety of wild bird species. Oral LD$_{50}$ values ranging from 100 to 1,000 mg/kg are reported for a variety of animals, including rodents, goats, sheep, and chickens.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 2.4 µg/liter
Chronic toxicity: 0.0043 µg/liter

Saltwater

Acute toxicity: 0.09 µg/liter
Chronic toxicity: 0.0040 µg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of chlordane in water are:

Risk	Concentration
10^{-5}	4.6 ng/liter
10^{-6}	0.46 ng/liter
10^{-7}	0.046 ng/liter

CAG Unit Risk (USEPA): 1.6 $(mg/kg/day)^{-1}$

OSHA Standard (skin): 0.5 mg/m^3 TWA

ACGIH Threshold Limit Values (skin): 0.5 mg/m^3 TWA
2 mg/m^3 STEL

Department of Transportation: Combustible liquid

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CHLORINE

Summary

Chlorine is a volatile gas that reacts in the atmosphere to produce hydrochloric acid, a strong acid. It is very reactive and therefore is not persistent in the environment. Chlorine gas is a strong irritant, and exposure to high concentrations will damage the lungs. Chlorine, measured as either total residual chlorine or chlorine-produced oxidants, is quite toxic to aquatic organisms.

CAS Number: 7782-50-5

Chemical Formula: Cl

Chemical and Physical Properties

Atomic Weight: 35.453

Boiling Point: -34.6°C

Melting Point: -100.98°C

Specific Gravity: 1.41 (liquid at 20°C)

Solubility in Water: Soluble (7.3 g/liter at 20°C)

Vapor Pressure: 4,800 mm Hg at 20°C

Vapor Density: 2.49

Transport and Fate

Volatilization of chlorine from aquatic or terrestrial systems can occur. In the atmosphere, chlorine can react with hydrocarbons to produce HCl, which can return to the earth with precipitation. Some researchers suggest that chlorine atoms can act as a catalyst in the degradation of the stratospheric ozone layer.

In water, chlorine reacts quickly to form hypochlorous acid (HOCl), which is weakly dissociated, and HCl. Depending on the pH level, HOCl, OCl^-, Cl^-, or Cl_2 may predominate in aqueous systems. Chlorine readily reacts with many types of organic matter and oxidizable inorganic matter. Chloroform

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and other chlorinated hydrocarbons are known to be formed as a result of the reaction of chlorine with humic substances and other organic materials. The presence of ammonia or amines along with chlorine can result in the formation of chloramines. These compounds generally are much more persistent than chlorine, hypochlorite, and many chlorinated hydrocarbons. In freshwater, the combination of combined (chloramines) and free chlorine is "total residual chlorine." In saltwater, several other chlorination products are also included, and chlorine levels are reported as "chlorine produced oxidants."

Health Effects

There are no reports of carcinogenic, teratogenic, or reproductive effects due to chlorine exposure in humans or experimental animals. One study reported the occurrence of chromosomal aberrations in cultured human lymphocytes after exposure to chlorine at 60 mg/m^3.

As a gas, chlorine is extremely irritating to the mucous membranes of the eyes and respiratory tract. Acute inhalation exposure to relatively high concentrations can damage the lungs and result in decreased lung capacity, pulmonary congestion, edema, and sometimes death. Other signs and symptoms include dyspnea and cough, cyanosis, corrosion of the teeth, severe headache, nausea, and syncope. Experiments with animals confirm the occurrence of irritant effects and lung damage as a result of acute or chronic exposure to chlorine. One-hour inhalation LC_{50} values of 879 mg/m^3 and 400 mg/m^3 are reported for the rat and mouse, respectively.

Toxicity to Wildlife and Domestic Animals

The acute toxicity of chlorine, measured as total residual chlorine (TRC) in freshwater and as chlorine produced oxidants (CPO) in saltwater, ranged from 17 µg/liter to 710 µg/liter for 31 freshwater species and from 25 µg/liter to 1,418 µg/liter for 23 saltwater species. Fish and invertebrate species generally had comparable ranges of sensitivity. Chronic studies have been conducted on 3 freshwater species and 1 saltwater species. Chronic values for the freshwater organisms ranged from 5 to 17 µg/liter, with acute-chronic ratios from 4 to 20. Chronic effects occurred in the saltwater organism at 47 µg/liter, and the acute-chronic ratio for this species was 1.2.

No information on the toxicity of chlorine to terrestrial wildlife or domestic animals was found in the literature reviewed.

Regulations and Standards

Proposed Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 14 μg/liter
Chronic toxicity: 8.3 μg/liter

Saltwater

Acute toxicity: 13 μg/liter
Chronic toxicity: 7.4 μg/liter

NIOSH Recommended Standard: 1.5 ppm

OSHA Standard: 3 mg/m^3

ACGIH Threshold Limit Values: 3 mg/m^3 TWA
9 mg/m^3 STEL

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CHLOROBENZENE

Summary

Chlorobenzene is used as a solvent and as a raw material in chemical manufacturing. It is persistent in the environment and can be adsorbed to organic material in soil. Chlorobenzene may cause liver tumors in male mice. Animals exposed to chlorobenzene have exhibited liver and kidney damage. Chlorobenzene is not very toxic to aquatic organisms; none of the LC_{50} values are less than 10 mg/liter.

CAS Number: 108-90-7

Chemical Formula: C_6H_5Cl

IUPAC Name: Chlorobenzene

Important Synonyms and Trade Names: Monochlorobenzene, benzene chloride, phenyl chloride

Chemical and Physical Properties

Molecular Weight: 112.6

Boiling Point: 131°C

Melting Point: -46°C

Specific Gravity: 1.11 at 20°C (liquid)

Solubility in Water: 500 mg/liter

Solubility in Organics: Soluble in alcohol, benzene, chloroform, ether, and carbon tetrachloride

Log Octanol/Water Partition Coefficient: 2.83

Vapor Pressure: 8.8 mm Hg at 20°C

Vapor Density: 3.88

Henry's Law Constant: 3.56×10^{-3} atm m^3/mole at 25°C

Flash Point: 28°C

Transport and Fate

Chlorobenzene is probably removed from surface water primarily by volatilization, although adsorption and bioaccumulation may also be factors. Monochlorobenzene would be expected to move slowly in soil because of its high octanol/water partition coefficient and consequent adsorption to soil organic material.

Health Effects

A study of the carcinogenicity of chlorobenzene was recently completed by the National Toxicology Program and preliminary results show that chlorobenzene caused neoplastic nodules in the liver of male rats but was not carcinogenic in female rats or in mice.

Occupational studies suggest that chronic exposure to monochlorobenzene vapor may cause blood dyscrasia, hyperlipidemia, and cardiac dysfunction in humans. Like many organic solvents, monochlorobenzene is a central nervous system depressant in overexposed humans, but no chronic neurotoxic effects have been reported. Animals exposed to chlorobenzene have exhibited liver and kidney damage and atrophy of the seminiferous tubules in the testes. The oral LD_{50} value for rats was 2910 mg/kg.

Toxicity to Wildlife and Domestic Animals

Chlorobenzene was acutely toxic to fish at levels greater than 25 mg/liter and to aquatic invertebrates at levels greater than 10 mg/liter. No chronic studies on the toxicity of chlorobenzene to aquatic life were found in the literature reviewed. Monochlorobenzene was shown to have a bioaccumulation factor of about 1,000 in freshwater species. No studies on terrestrial wildlife or domestic animals were reported in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Health criterion: 488 μg/liter
Organoleptic criterion: 20 μg/liter

OSHA Standard (air): 350 mg/m^3 TWA

ACGIH Threshold Limit Value: 350 mg/m^3 TWA

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CHLOROBENZILATE

Summary

Chlorobenzilate is an organochlorine pesticide used to control mites. It is moderately persistent in the environment. Chronic ingestion of chlorobenzilate caused testicular atrophy in male rats, enlarged livers in female rats and liver cancer in several strains of mice. It is moderately toxic to aquatic organisms, with acute toxicity values as low as 550 μg/liter.

CAS Number: 510-15-6

Chemical Formula: $C_{16}H_{14}Cl_2O_3$

IUPAC Name: Ethyl-4,4-dichlorobenzilate

Important Synonyms and Trade Names: Akar, Benzilan, Folbex

Chemical and Physical Properties

Molecular Weight: 325.2

Boiling Point: 141-142°C at 0.06 mm Hg

Melting Point: 35-37°C

Specific Gravity: 1.2816 at 20°C

Solubility in Water: Practically insoluble

Solubility in Organics: Soluble in most organic solvents and petroleum oils

Log Octanol/Water Partition Coefficient: Approximately 5 (calculated)

Vapor Pressure: $6.8x10^{-6}$ mm Hg at 20°C

Transport and Fate

Little information on the transport and fate of chlorobenzilate was found in the sources reviewed. However, some generalizations can be made based on its chemical and physical properties and on the information available about related chemicals. Chlorobenzilate appears to be moderately persistent. Although it has a low vapor pressure, as in the case of other organochloride compounds, volatilization is probably an important

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transport process. The high log octanol/water partition coefficient indicates that chlorobenzilate is probably readily sorbed by soil materials and sediment and will not move easily through groundwater or surface water. Chlorobenzilate has been shown to be metabolized in dogs and may therefore be biodegraded by other organisms. Based on this information and data on the degradation of DDT, soil bacteria may play an important role in the fate of chlorobenzilate.

Health Effects

Chlorobenzilate produced hepatocellular carcinomas in both males and females in one strain of mice (NCI 1978) and in male mice in two other strains (IARC 1983). A slightly increased incidence of adrenocortical adenomas was seen in rats of both sexes, but these data were considered inadequate for evaluation by IARC (1983). Chlorobenzilate does not appear to be mutagenic and did not adversely affect reproduction in a three-generation study.

Female rats fed 100 ppm chlorobenzilate (approximately 5 mg/kg bw/day) for 4 weeks developed enlarged livers. Male rats fed 1,600 or 3,000 ppm of chlorobenzilate (approximately 135 mg/kg bw/day) for 78 weeks experienced testicular atrophy. The acute oral LD_{50} value for rats, mice, and hamsters was 700 mg/kg.

Toxicity to Wildlife and Domestic Animals

Rainbow trout exposed to chlorobenzilate for 48 hours had an LC_{50} value of 710 μg/liter. A 48-hour LC_{50} of 550 μg/liter was determined for water fleas. No other information on the toxicity of chlorobenzilate to wildlife or domestic animals was found in the literature reviewed.

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WORTHING, C.R., ed. 1979. The Pesticide Manual: A World Compendium. British Crop Protection Council, Croydon, England. 655 pages

CHLOROETHANE

Summary

Chloroethane is used as a solvent, as a refrigerant, and as a raw material in the manufacture of tetraethyl lead. It is fairly volatile in the environment. Chloroethane caused headaches and dizziness in workers exposed to high levels. It causes kidney damage and liver changes in chronically exposed animals.

CAS Number: 75-00-3

Chemical Formula: C_2H_5Cl

IUPAC Name: Chloroethane

Important Synonyms and Trade Names: Ethyl chloride, monochloroethane

Chemical and Physical Properties

Molecular Weight: 64.52

Boiling Point: 12.3°C

Melting Point: -136.4°C

Specific Gravity: 0.8978 at 20°C

Solubility in Water: 5740 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol and ether

Log Octanol/Water Partition Coefficient: 1.54

Vapor Pressure: 1,000 mm Hg at 20°C

Vapor Density: 2.23

Transport and Fate

Chloroethane is probably not very persistent in the environment. It volatilizes rapidly from water; once in the atmosphere, it is photooxidized, and formyl chloride is the initial oxidation product. Hydrolysis may also occur in surface water or in moist soil. Biodegradation, sorption, and bioaccumulation probably are not important fate processes for chloroethane.

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Health Effects

Chloroethane is presently being tested by the National Toxicology Program (NTP) for carcinogenicity and genetic toxicity. No information evaluating its reproductive toxicity or teratogenicity was found. Chloroethane caused minor neurological effects (e.g., headache, dizziness) in workers exposed to high levels. In animals, chronic exposure to chloroethane caused kidney damage and fatty changes in the liver, and at high levels upset cardiac rhythm. Monochloroethane is considered to be the least toxic of the chlorinated ethanes.

Toxicity to Wildlife and Domestic Animals

No information was found on the toxicity of chloroethane to wildlife or domestic animals. The toxicity of other chlorinated ethanes to aquatic organisms generally declines with decreasing chlorine content. Therefore, chloroethane is probably less toxic than 1,2-dichloroethane, which causes acute toxicity at about 120 mg/liter and chronic toxicity at 20 mg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data were not adequate for establishing criteria.

OSHA Standard (air): 2,600 mg/m^3 TWA

ACGIH Threshold Limit Values: 2,600 mg/m^3 TWA
3,250 mg/m^3 STEL

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BIS(2-CHLOROETHOXY)ETHANE

Summary

bis(2-Chloroethoxy)ethane is probably somewhat persistent in the environment. It has oral LD_{50} values in rats and guinea pigs of 250 mg/kg and 120 mg/kg, respectively.

CAS Number: 112-26-5

Chemical Formula: $C_6H_{12}Cl_2O_2$

IUPAC Name: 1,2-bis-2-Chloroethoxyethane

Important Synonyms and Trade Names: Triglycol dichloride, triethylene glycol dichloride

Chemical and Physical Properties

Molecular Weight: 187

Boiling Point: 241.3°C

Melting Point: -31.5°C

Specific Gravity: 1.2 at 20°C

Solubility in Water: Approximately 5,000 mg/liter (calculated)

Log Octanol/Water Partition Coefficient: 1.92 (calculated)

Vapor Pressure: Probably less than 0.1 mm Hg at 20°C

Flash Point: 121°C

Transport and Fate

No information is available on the transport and fate of bis(2-chloroethoxy)ethane. However, this information can be extrapolated from data on bis(2-chloroethoxy)methane data and from the chemical and physical properties of bis(2-chloroethoxy)ethane.

bis(2-Chloroethoxy)ethane is probably rather persistent in the environment. It has a low vapor pressure and therefore probably is not very volatile. Its calculated log octanol/water partition coefficient (1.92) and solubility suggest that it may leach through the soil if it is not biodegraded. There is no

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information on the biodegradation of bis(2-chloroethoxy)ethane. Based on information for bis(2-chloroethoxy)methane, the most likely fate processes for bis(2-chloroethoxy)ethane are slow hydrolysis and oxidation to peroxides.

Health Effects

Limited information is available on the health effects of bis(2-chloroethoxy)ethane. Three acute studies indicated that the chemical had oral LD_{50} values of 250 mg/kg in rats and 120 mg/kg in guinea pigs and an LD_{50} of 1,410 mg/kg when applied to the skin of mice.

More information on the potential effects of bis(2-chloroethoxy) ethane can be inferred from studies on bis(2-chloromethoxy)ethane. This chemical produced local sarcomas when applied dermally, by subcutaneous injection, or intraperitoneally.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of bis(2-chloroethoxy)ethane to wildlife or domestic animals was found in the sources reviewed.

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BIS(2-CHLOROETHYL)ETHER

Summary

bis(2-Chloroethyl)ether was used in the past as a soil fumigant and is now used as a solvent and chemical reagent. It is fairly soluble in water and is probably moderately persistent in the environment. bis(2-Chloroethyl)ether caused an increased incidence of liver tumors in male mice following oral administration, and it was found to be mutagenic using the Ames assay. In the air, it is irritating to the eyes and nasal passages and when inhaled can damage the lungs, liver, kidneys, and brain.

CAS Number: 111-44-4

Chemical Formula: $ClCH_2CH_2OCH_2CH_2Cl$

IUPAC Name: bis(beta-Chloroethyl)ether

Important Synonyms and Trade Names: sym-Dichloroethyl ether; 2,2'-Dichloroethyl ether; 1-Chloro-2-(beta-chloroethoxy)-ethane; DCEE; 1,1'-oxybis-(2-chloroethane)

Chemical and Physical Properties

Molecular Weight: 143.02

Boiling Point: 178°C

Melting Point: -24.5°C

Specific Gravity: 1.22 at 20°C

Solubility in Water: 10,200 mg/liter

Solubility in Organics: Miscible with most organic solvents

Log Octanol/Water Partition Coefficient: 1.58

Vapor Pressure: 0.71 mm Hg at 20°C

Vapor Density: 4.93

Flash Point: 55°C

Transport and Fate

There is little information available concerning the environmental transport and fate of bis(2-chloroethyl)ether and the relative importance of the various transport and fate processes. Some volatilization of this compound from aquatic and terrestrial systems, and subsequent atmospheric transport probably can occur. Because it is somewhat soluble in water, bis(2-chloroethyl)-ether can migrate through the soil. Direct photolysis is not expected to take place in the atmosphere or in surface waters. However, photo oxidation of the bis(2-chloroethyl)ether that reaches the troposphere is likely to occur. Slow hydrolytic cleavage of the carbon-chlorine bonds can occur and is probably the most important aquatic fate.

Adsorption on particulate matter does not appear to be a significant environmental transport process. A limited amount of indirect evidence suggests that bis(2-chloroethyl)ether has little potential for bioaccumulation. Available information is not adequate to characterize the importance of biodegradation as a fate process. It is reported that significant degradation can occur in aquatic systems after a period of acclimation.

Health Effects

bis(2-Chloroethyl)ether caused an increased incidence of hepatomas in male mice following oral administration. It is also reported to be mutagenic in Salmonella tester strains. No data concerning teratogenic or reproductive effects are available.

bis(2-Chloroethyl)ether concentrations of 100 ppm (600 mg/m^3) and possibly lower are irritating to the eyes and nasal passages, and may cause coughing and nausea. Exposure to concentrations above 550 ppm (3,300 mg/m^3) is considered to be intolerable. Concentrations of 500 ppm and 250 ppm are reported to be fatal in guinea pigs and rats, respectively. The most severe toxic effects are seen in the lungs, although the kidneys, liver, and brain may also be affected. No serious toxic effects were noted following chronic exposure of guinea pigs and rats to 69 ppm (420 mg/m^3) of bis(2-chloroethyl)ether.

bis(2-Chloroethyl)ether is a mild skin irritant. However, acutely toxic and lethal amounts may be absorbed through the skin. An oral LD_{50} of 75 mg/kg is reported for the rat.

Toxicity to Wildlife and Domestic Animals

Data adequate to characterize the toxicity of bis(2-chloroethyl)ether to wildlife and domestic animals are not available.

Acute toxicity of chloroalkyl ethers, in general, to freshwater aquatic life is reported to occur at concentrations as low as 238,000 μg/liter and would occur at lower concentrations among species more sensitive than those tested.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of bis(2-chloroethyl)ether in water are:

Risk	Concentration
10^{-5}	0.3 μg/liter
10^{-6}	0.03 μg/liter
10^{-7}	0.003 μg/liter

CAG Unit Risk (USEPA): 1.14 $(mg/kg/day)^{-1}$

OSHA Standard: 90 mg/m^3 Ceiling Level

ACGIH Threshold Limit Values: 30 mg/m^3 TLV
60 mg/m^3 STEL

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CHLOROFORM

Summary

Chloroform (trichloromethane) is often produced during the chlorination of drinking water and thus is a common drinking water contaminant. It is volatile in surface waters and is not likely to be persistent in the environment. Chloroform caused an increase in kidney epithelial tumors in rats and in hepatocellular carcinomas in mice. In addition, there is suggestive evidence from epidemiological studies that exposure to chloroform and other trihalomethanes is associated with an increased incidence of bladder tumors in humans. Other toxic effects of chloroform include central nervous system depression; eye, skin, and gastrointestinal irritation; and damage to the liver, heart, and kidney.

CAS Number: 67-66-3

Chemical Formula: $CHCl_3$

IUPAC Name: Trichloromethane

Chemical and Physical Properties

Molecular Weight: 119.38

Boiling Point: 61.7°C

Melting Point: -63.5°C

Specific Gravity: 1.4832 at 20°C

Solubility in Water: 8,200 mg/liter at 20°C

Solubility in Organics: Soluble in acetone; miscible with alcohol, ether, benzene, and ligroin

Log Octanol/Water Partition Coefficient: 1.97

Vapor Pressure: 150.5 mm Hg at 20°C

Vapor Density: 4.12

Transport and Fate

Volatilization into the atmosphere is the major transport process for removal of chloroform from aquatic systems (USEPA 1979). Once in the troposphere, chloroform is attacked by hydroxyl radicals with the subsequent formation of phosgene ($COCl_2$) and possibly chlorine oxide (ClO) radicals. Neither of these reaction products is likely to persist; phosgene is readily hydrolyzed to hydrochloric acid and carbon dioxide. Reaction with hydroxy radicals is thought to be the primary environmental fate of chloroform. However, chloroform that remains in the troposphere may return to earth in precipitation or adsorbed on particulates, and a small amount may diffuse upward to the stratosphere where it photodissociates via interaction with ultraviolet light.

Photolysis, hydrolysis, and sorption do not appear to be significant environmental fate processes for chloroform. However, sorption processes may have some importance as a removal mechanism in groundwater and soil. The log octanol/water partition coefficient indicates that this compound may bioaccumulate under conditions of constant exposure. Studies with marine organisms provide evidence for only weak to moderate bioaccumulation. Although chloroform is somewhat lipophilic and tends to be found at higher concentrations in fatty tissues, there is no evidence for biomagnification in aquatic food chains.

Health Effects

Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence of hepatocellular carcinomas in mice (IARC 1979, USEPA 1980). Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with an increased frequency of bladder cancer in humans. However, these results are not sufficient to establish causality. An increased incidence of fetal abnormalities was reported in offspring of pregnant rats exposed to chloroform by inhalation. Oral doses of chloroform that caused maternal toxicity produced relatively mild fetal toxicity in the form of reduced birth weights. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Toxic effects include local irritation of the skin or eyes, central nervous system depression, gastrointestinal irritation, liver and kidney damage, cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from chloroform overdosing can occur and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis.

Exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produces liver and kidney damage in laboratory animals. The oral LD$_{50}$ and inhalation LC$_{LO}$ values for the rat are 908 mg/kg and 39,000 mg/m^3 per 4 hours, respectively (ACGIH 1980).

Toxicity to Wildlife and Domestic Animals

Limited information is available concerning the toxicity of chloroform to organisms exposed at known concentrations (USEPA 1980). Median effect concentrations for two freshwater and one invertebrate species range from 28,900 to 115,000 µg/liter. Twenty-seven day LC$_{50}$ values of 2,030 and 1,240 µg/liter were reported for embryo-larval tests with rainbow trout in water at two levels of hardness. The only reliable result concerning the toxicity of chloroform to saltwater aquatic life is a 96-hour LC$_{50}$ value of 81,500 µg/liter for pink shrimp.

An equilibrium bioconcentration factor of six with a tissue half-life of less than 1 day was determined for the bluegill. Although chloroform is not strongly bioaccumulated, it is thought to be widely distributed in the environment and can be detected in fish, water birds, marine mammals, and various crops.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of chloroform in water are:

Risk	Concentration
10^{-5}	1.90 µg/liter
10^{-6}	0.19 µg/liter
10^{-7}	0.019 µg/liter

CAG Unit Risk (USEPA): $8.1 \times 10^{-2} (mg/kg/day)^{-1}$

Primary Drinking Water Standard: 0.10 mg/liter (total trihalomethanes)

NIOSH Recommended Standard: 9.8 mg/m^3 1-hr Ceiling Level

OSHA Standard: 244 mg/m^3 Ceiling Level

ACGIH Threshold Limit Value: 50 mg/m^3 (suspected human carcinogen)

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p-CHLORO-m-CRESOL

Summary

p-Chloro-m-cresol is moderately soluble in water and is readily degraded during aerobic sewage treatment. However, it is probably moderately persistent in the natural environment. p-Chloro-m-cresol causes dermatitis and natural allergic reactions in sensitive individuals. It causes kidney damage in mice. p-Chloro-m-cresol was acutely toxic to fathead minnows at concentrations of 30 μg/liter.

CAS Number: 59-50-7

Chemical Formula: $C_6H_3CH_3ClOH$

IUPAC Name: 4-Chloro-m-cresol

Important Synonyms and Trade Names: 4-Chloro-3-methylphenol,
3-chloro-5-hydroxytoluene,
3-methyl-4-chlorophenol

Chemical and Physical Properties

Molecular Weight: 142.59

Boiling Point: 253°C

Melting Point: 66-68°C

Specific Gravity: 1.215 at 15°C

Solubility in Water: 3,850 mg/liter

Solubility in Organics: Soluble in alcohol and ether

Log Octanol/Water Partition Coefficient: 2.95 (calculated)

Transport and Fate

Experimental evidence with related compounds and theoretical considerations suggest that intramolecular photolysis is the most likely environmental fate for p-chloro-m-cresol. These reactions could produce a mixture of compounds from initial intermediates in which the methyl group becomes chlorinated or becomes oxidized to a benzyl hydroperoxide. Although little information concerning other environmental processes is available, it appears that oxidation and hydrolysis are not likely to be important fates, and that volatilization and sorption are not likely to be important transport processes.

The log octanol/water partition coefficient of p-chloro-m-cresol suggests that it may have a tendency to bioaccumulate, but that it is probably not an important fate process. Para-chloro-m-cresol is readily degraded during aerobic sewage treatment and is partially degraded by adapted mixed cultures of soil and water microorganisms. However, the potential for biodegradation in ambient surface waters or in soil is unknown.

Health Effects

No information concerning the carcinogenicity, mutagenicity, or teratogenicity of p-chloro-m-cresol in humans or experimental animals is available. Para-chloro-m-cresol, in a 1.5% aqueous solution, is reported to produce pruritic vesicular dermatitis in sensitive individuals. Systemic reactions to mucous heparin preserved with 0.15% of an unspecified chlorocresol, likely to be p-chloro-m-cresol, include collapse, pallor, sweating, hypotension, tachycardia, and generalized urticarial rash. In another case, severe burning pain occurred at the site of injection with heparin preserved with 0.15% chlorocresol. Shortly afterwards, nausea, lightheadedness, and drowsiness accompanied by pallor and sweating appeared.

Intravenous or subcutaneous administration of p-chloro-m-cresol produced severe muscle tremors and death in mice and rats. Damage to renal tubules was also observed. In the mouse, the reported intravenous and subcutaneous LD_{50} values are both 70 mg/kg. A subcutaneous LD_{50} of 400 mg/kg and an oral LD_{50} of 500 mg/kg are reported for the rat.

Toxicity to Wildlife and Domestic Animals

An acute toxicity value of 30 µg/liter is reported for the fathead minnow, a freshwater species. An LC_{50} value due to chlorosis of 95,488 µg/liter is reported for duckweed, a freshwater plant species.

No other information concerning the toxicity of p-chloro-m-cresol to terrestrial wildlife and domestic animals is available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Organoleptic criterion: 3,000 μg/liter

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Summary

1-Chloro-3-nitrobenzene is used in the manufacture of dyes. The limited information available on the transport and fate of the chemical suggests that biodegradation is an important fate process, but one that occurs slowly. Consequently, it is probably moderately persistent in the environment. 1-Chloro-3-nitrobenzene was mutagenic in the Ames assay. It causes methemoglobinemia in experimental animals and was reported to induce hemolytic activity in rats.

CAS Number: 121-73-3

Chemical Formula: $C_6H_4ClNO_2$

IUPAC Name: 1-Chloro-3-nitrobenzene

Important Synonyms and Trade Names: Chloro-m-nitrobenzene, m-chloronitrobenzene, nitrochlorobenzene

Chemical and Physical Properties

Molecular Weight: 157.56

Boiling Point: 235-236°C

Melting Point: 46°C

Specific Gravity: 1.534 at 20°C

Solubility in Water: Insoluble in water

Solubility in Organics: Soluble in alcohol, ether, and carbon disulfide

Log Octanol/Water Partition Coefficient: 2.43

Transport and Fate

The only information on the transport and fate of 1-chloro-3-nitrobenzene indicates that biodegradation by soil bacteria is an important fate process but that it occurs slowly. Based on this data, on information for nitrobenzene and chlorobenzenes, and on the chemical and physical properties of the compound, it would appear that 1-chloro-3-nitrobenzene is quite persistent in the environment. Besides biodegradation, other potential

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if the compound is adsorbed to humic material near the soil or water surface.

Health Effects

1-Chloro-3-nitrobenzene has not been tested for carcinogenicity in animal bioassays. However, both 1-chloro-4-nitrobenzene and 1-chloro-2-nitrobenzene were reported to be carcinogenic in mice (Weisburger et al. 1978). 1-Chloro-3-nitrobenzene was found to be mutagenic using the Ames assay without metabolic activation in strain TA100. No information was available on the teratogenicity, embryotoxicity, or fetotoxicity of 1-chloro-3-nitrobenzene.

It has been reported that 1-chloro-3-nitrobenzene causes methemoglobunemia in experimental animals. It led to the formation of sulfhemoglobin in rats and was reported to have a hemolytic action, with resulting anemia and cyanosis. 1-Chloro-3-nitrobenzene is probably reduced to chloroaniline in the body.

The oral LD_{50} in the rat is 470 mg/kg, and the mouse oral LD_{50} is 380 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of 1-chloro-3-nitrobenzene to wildlife or domestic animals was available in the literature reviewed.

Regulations and Standards

No regulations or standards have been established for 1-chloro-3-nitrobenzene.

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Summary

Chromium is a heavy metal that generally exists in either a trivalent or hexavalent oxidation state. Hexavalent chromium (Cr VI) is rather soluble and is quite mobile in groundwater and surface water. However, in the presence of reducing agents it is rapidly converted to trivalent chromium (Cr III), which is strongly adsorbed to soil components and consequently is much less mobile. A number of salts of hexavalent chromium are carcinogenic in rats. In addition, an increased incidence of lung cancer was seen in workers occupationally exposed to chromium VI. Hexavalent chromium also causes kidney damage in animals and humans. Trivalent chromium is less toxic than hexavalent chromium; its main effect is contact dermatitis in sensitive individuals.

CAS Number: 7440-47-3

Chemical Formula: Cr

IUPAC Name: Chromium

Chemical and Physical Properties (Metal)

Atomic Weight: 51.996

Boiling Point: 2672°C

Melting Point: 1857 ± 20°C

Specific Gravity: 7.20 at 28°C

Solubility in Water: Insoluble; some compounds are soluble

Transport and Fate

Hexavalent Cr is quite soluble, existing in solution as a component of a complex anion. It is not sorbed to any significant degree by clays or hydrous metal oxides. The anionic form varies according to pH and may be a chromate, hydrochromate, or dichromate. Because all anionic forms are so soluble, they are quite mobile in the aquatic environment. Cr VI is efficiently

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removed by activated carbon and thus may have some affinity for organic materials in natural water. Cr VI is a moderately strong oxidizing agent and reacts with reducing materials to form trivalent chromium. Most Cr III in the aquatic environment is hydrolyzed and precipitates as chromium hydroxide. Sorption to sediments and bioaccumulation will remove much of the remaining Cr III from solution. Cr III is adsorbed only weakly to inorganic materials. Cr III and Cr VI are readily interconvertible in nature depending on microenvironmental conditions such as pH, hardness, and the types of other compounds present. Soluble forms of chromium accumulate if ambient conditions favor Cr VI. Conditions favorable for conversion to Cr III lead to precipitation and adsorption of chromium in sediments.

In air, chromium is associated almost entirely with particulate matter. Sources of chromium in air include windblown soil and particulate emissions from industrial processes. Little information is available concerning the relative amounts of Cr III and Cr VI in various aerosols. Relatively small particles can form stable aerosols and can be transported many miles before settling out.

Cr III tends to be adsorbed strongly onto clay particles and organic particulate matter, but can be mobilized if it is complexed with organic molecules. Cr III present in minerals is mobilized to different extents depending on the weatherability and solubility of the mineral in which it is contained. Hexavalent compounds are not strongly adsorbed by soil components and Cr VI is mobile in groundwater. Cr VI is quickly reduced to CR III in poorly drained soils having a high content of organic matter. Cr VI of natural origin is rarely found in soils.

Health Effects

The hexavalent form of chromium is of major toxicological importance in higher organisms. A variety of chromate (Cr VI) salts are carcinogenic in rats and an excess of lung cancer has been observed among workers in the chromate-producing industry. Cr VI compounds can cause DNA and chromsome damage in animals and humans, and Cr (VI) trioxide is teratogenic in the hamster. Inhalation of hexavalent chromium salts causes irritation and inflammation of the nasal mucosa, and ulceration and perforation of the nasal septum. Cr VI also produces kidney damage in animals and humans. The liver is also sensitive to the toxic effects of hexavalent Cr, but apparently less so than the kidneys or respiratory system. Cr III is less toxic than Cr VI; its main effect in humans is a form of contact dermatitis in sensitive individuals.

Toxicity to Wildlife and Domestic Animals

Chromium is an essential nutrient and is accumulated in a variety of aquatic and marine biota, especially benthic organisms, to levels much higher than in ambient water. Levels in biota, however, usually are lower than levels in the sediments. Passage of chromium through the food chain can be demonstrated. The food chain appears to be a more efficient pathway for chromium uptake than direct uptake from seawater.

Water hardness, temperature, dissolved oxygen, species, and age of the test organism all modify the toxic effects of chromium on aquatic life. Cr III appears to be more acutely toxic to fish than Cr VI; the reverse is true in long term chronic exposure studies.

None of the plants normally used as food or animal feed are chromium accumulators. Chromium absorbed by plants tends to remain primarily in the roots and is poorly translocated to the leaves. There is little tendency for chromium to accumulate along food chains in the trivalent inorganic form. Organic chromium compounds, about which little is known, can have significantly different bioaccumulation tendencies. Little information concerning the toxic effects of chromium on mammalian wildlife and domestic animal species is available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Cr VI:

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity: 11 μg/liter
Chronic toxicity: 7.2 μg/liter

Saltwater

Acute toxicity: 1,200 μg/liter
Chronic toxicity: 54 μg/liter

Human Health

Criterion: 50 μg/liter

Cr III:

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity: $e^{(0.819[\ln(\text{hardness})]+3.568)}$ µg/liter

Chronic toxicity: $e^{(0.819\ [\ln(\text{hardness})])+0.537)}$ µg/liter

Saltwater

The available data are not adequate for establishing criteria.

Human Health

Criterion: 170 mg/liter

CAG Unit Risk for inhalation exposure to CR VI (USEPA): 41 $(\text{mg/kg/day})^{-1}$

National Interim Primary Drinking Water Standard: 50 µg/liter

NIOSH Recommended Standards for CR VI: $1\ \mu g/m^3$ carcinogenic
$25\ \mu g/m^3$ noncarcinogenic TWA
$50\ \mu g/m^3$ noncarcinogenic (15-min sample)

OSHA Standards: OSHA air standards have been set for several chromium compounds. Most recognized or suspected carcinogenic chromium compounds have ceiling limits of $100\ \mu g/m^3$.

ACGIH Threshold Limit Values: Several chromium compounds have TWAs ranging from 0.05 to $0.5\ mg/m^3$. Chromite ore processing (chromate), certain water insoluble Cr VI compounds, and chromates of lead and zinc are recognized or suspected human carcinogens and have $0.05\ mg/m^3$ TWAs.

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CHRYSENE

Summary

Chrysene is a five-ringed polycyclic aromatic hydrocarbon (PAH). It is rather persistent in the environment; biodegradation is probably the ultimate fate process. Dermal application of chrysene produces skin tumors in mice, and subcutaneous injection produces local sarcomas. Chrysene was found to be mutagenic using several test systems. Although there is little information on other toxic effects of chrysene, carcinogenic PAHs as a group cause skin disorders and have an immunosuppressive effect.

CAS Number: 218-01-9

Chemical Formula: $C_{18}H_{12}$

IUPAC Name: Chrysene

Important Synonyms and Trade Names: 1,2-Benzophenanthrene; benz(a)phenanthrene

Chemical and Physical Properties

Molecular Weight: 228.28

Boiling Point: 448°C

Melting Point: 256°C

Specific Gravity: 1.274 at 20°C

Solubility in Water: 0.002 mg/liter at 25°C

Solubility in Organics: Soluble in ether, alcohol, glacial and acetic acid

Log Octanol/Water Partition Coefficient: 5.61

Vapor Pressure: 10^{-11} to 10^{-6} mm Hg at 20°C

Transport and Fate

Very little specific information concerning the environmental transport and fate of chrysene is available. However, data can be derived with reasonable confidence from information concerning benzo(a)anthracene and other related polycyclic aromatic hydrocarbons (PAHs). Dissolved chrysene may undergo rapid, direct photolysis in aquatic systems. However, the relative importance of this process as an environmental fate is unknown. Singlet oxygen is the oxidant and quinones are the products in photolysis reactions involving polycyclic aromatic hydrocarbons. Free-radical oxidation of chrysene is likely to be slow and is not likely to be a significant fate process. Because chrysene does not contain groups amenable to hydrolysis, this process is not thought to be a significant environmental fate. Volatilization does not appear to be an important transport process.

Chrysene probably accumulates in the sediment and biota portions of the aquatic environment, and adsorption to suspended matter is likely to be the dominant transport process. It is probable that sorption onto sediments, soil particles, and biota is strongly correlated with the organic carbon levels present. Bioaccumulation of chrysene is expected to be short term and is not an important fate process. Although polycyclic aromatic hydrocarbons with four or less aromatic rings, like chrysene, are readily and quickly bioaccumulated, they also are rapidly metabolized and excreted. These kinds of PAHs are degraded by microbes and readily metabolized by multicellular organisms. Degradation by mammals is considered to be incomplete; the parent compound and metabolites are excreted by the urinary system. Biodegradation is probably the ultimate fate process for chrysene. However, the speed and extent of this process are unknown. Biodegradation of PAHs generally occurs more rapidly in soil than in aquatic systems and is also faster in those systems chronically contaminated with these compounds.

Atmospheric transport of chrysene can occur, and chrysene can be returned to aquatic and terrestrial systems by atmospheric fallout and with precipitation. It can enter surface and groundwaters by leaching from polluted soils.

Health Effects

The potential for polycyclic aromatic hydrocarbons to induce malignant transformation dominates the consideration given to health hazards resulting from exposure. This is because overt signs of toxicity are often not produced until the dose is sufficient to produce a high tumor incidence.

No case reports or epidemiological studies on the significance of chrysene exposure to humans are available. However, coal tar and other materials known to be carcinogenic to humans may contain chrysene. Chrysene produces skin tumors in mice following repeated dermal application. High subcutaneous doses are reported to result in a low incidence of tumors with a long induction time in mice. Chrysene is considered to have weak carcinogenic activity compared to benzo(a)pyrene. Chrysene is reported to be mutagenic in a variety of test systems. No information concerning the teratogenic effects of chrysene in humans or experimental animals is available.

Although there is little information concerning other toxic effects of chrysene, it is reported that applying the carcinogenic PAHs to mouse skin leads to the destruction of sebaceous glands, hyperplasia, hyperkeratosis, and ulceration. Workers exposed to materials containing these compounds may exhibit chronic dermatitis, hyperkeratoses, and other skin disorders. Although specific results with chrysene are not reported, it has been shown that many carcinogenic PAHs have an immunosuppressive effect.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of the toxicity of chrysene to domestic animals and wildlife are not available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

Risk	Concentration
10^{-5}	28 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

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COBALT

Summary

Cobalt generally occurs in the 0 or +2 oxidation states. Elemental cobalt is relatively unreactive and is quite stable in air or water. Cobalt caused injection site sarcomas in rats, but the results of other studies were negative; it is not considered to pose a carcinogenic risk to humans. Chronic oral exposure causes goiter, decreased thyroid function, increased heart and respiratory rates, and blood lipid changes. Cobalt causes respiratory disease among occupationally exposed workers.

CAS Number: 7440-48-4

Chemical Formula: Co

IUPAC Name: Cobalt

Chemical and Physical Properties (Metal)

Atomic Weight: 58.933

Boiling Point: 2,870°C

Melting Point: 1,495°C

Specific Gravity: 8.9

Solubility in Water: Insoluble; some salts are soluble

Transport and Fate

Very little cobalt appears to occur in soluble form in natural aquatic systems. Several surveys show that cobalt frequently is not detectable and that concentrations greater than 10 µg/liter are rare. The most important control on mobility of cobalt in aquatic and terrestrial systems is probably adsorption to the clay minerals and hydrous oxides of iron, manganese, and aluminum that are often present in the clay fractions of sediments and soils. The principal factors controlling adsorption and desorption processes are pH, Eh, and the concentrations of cobalt and competing compounds. Chelation of cobalt with some organic compounds can also occur. Small amounts of cobalt may be solubilized by bacteriological activity. Cobalt is an essential element and can be accumulated by plants and animals, though generally not to excessive concentrations. Photolysis, volatilization, and biotransformation are not important environmental fate processes for cobalt. However, some atmospheric transport of cobalt and cobalt compounds can occur.

Health Effects

Cobalt metal and cobalt oxide have been reported to cause injection site sarcomas in rats (Gilman 1962, Heath 1960). However, this type of response by itself is not generally considered adequate evidence of a chemical's carcinogenicity. The absence of positive carcinogenic responses in other studies with experimental animals and the lack of epidemiologic evidence suggest that cobalt and its compounds are unlikely to pose a carcinogenic risk to humans. Limited data indicate that cobalt chloride has mutagenic activity in a variety of test systems. This compound was also reported to cause craniofacial developmental abnormalities in the offspring of mice exposed by intraperitoneal injection during pregnancy. No other information indicating carcinogenic, mutagenic, or teratogenic activity is available.

Ingestion of excessive amounts of cobalt as a result of therapeutic administration was reported to produce vomiting, diarrhea, and a sensation of warmth in humans. A lethal dose of 1,500 mg/kg was reported for a child. Intravenous administration may cause flushing of the face, increased blood pressure, slowed respiration, giddiness, tinnitus, and deafness due to nerve damage. Chronic oral exposure to cobalt can cause goiter and decreased thyroid function, increased heart and respiration rates, and blood lipid changes. These effects were reported to occur in children receiving between 1 and 6 mg/kg per day as part of a treatment for anemia. The symptoms did not persist after cessation of therapy. Cobalt salts included in a beer formulation at concentrations of 1.2 to 1.5 mg/liter were reported to be responsible for a number of deaths due to congestive heart failure. Intake of this amount of cobalt is well below the amount that can normally be ingested safely by humans. However, studies with experimental animals show that ethanol potentiates the toxic effects of cobalt.

Some workers occupationally exposed to dust during the manufacture and use of tungsten carbide developed respiratory disease. Cobalt metal is currently thought to be the causative factor in these cases. Two types of disease developed: The first is a nonprogressive, asthma-like reaction that does not persist after cessation of exposure. The second is "hard metal disease." This disease is progressive, and after a certain stage, the changes in lung structure and function become irreversible, with death from cardiopulmonary insufficiency usually occurring.

The oral LD$_{50}$ value for cobalt is 1,500 mg/kg in the rat. The oral LD$_{50}$ values for a variety of inorganic cobalt compounds range from about 150 mg/kg for cobalt fluoride to 503 mg/kg for cobalt acetate (Speijers et al. 1982).

Toxicity to Wildlife and Domestic Animals

Little information regarding toxic effects of exposure to cobalt or cobalt compounds is available. Acute cobalt toxicity is seen in chickens at 50 ppm in the diet (approximately 3 mg/kg of body weight) per day and in sheep at 6 mg/kg of body weight per day. In sheep, daily doses of 3 mg/kg of body weight, which is about 1,000 times the normal daily intake of cobalt, do not produce harmful effects, even after several weeks.

Regulations and Standards

OSHA Standard: 0.1 mg/m^3 TWA

ACGIH Threshold Limit Value: 0.05 mg/m^3 TWA

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COPPER

Summary

Copper is among the more mobile metals in the environment. It is toxic to humans at high levels; it causes irritation following acute exposure and anemia following chronic exposure. Sheep are very susceptible to copper toxicosis, as are many aquatic organisms.

Background Information

Copper exists in a valence state of +1 or +2. It is a lustrous, reddish metal. The physical properties of copper include ductility and conductivity of heat and electricity. Copper is found in nature as sulfide, oxide, or carbonate ore.

CAS Number: 7440-50-8

Chemical Formula: Cu

IUPAC Name: Copper

Chemical and Physical Properties

Atomic Weight: 63.546

Boiling Point: 2,567°C

Melting Point: 1,083°C

Specific Gravity: 8.92

Solubility in Water: Most copper salts are insoluble, with the exception of $CuSO_4$, $Cu(NO_3)_2$, and $CuCl_2$ (the more common copper salts). The metal is insoluble in water.

Vapor Pressure: 1 mm Hg at 1,628°C

Transport and Fate

Copper has two oxidation states, +1 (cuprous) and +2 (cupric). Cuprous copper is unstable in aerated water over the pH range of most natural waters (6 to 8) and oxidizes to the cupric state. Several processes determine the fate of copper in the aquatic environment: formation of complexes, especially with humic substances; sorption to hydrous metal oxides, clays, and organic materials; and bioaccumulation. In waters polluted with soluble organic material, complexation with organic ligands can occur, thus favoring the prolonged dispersion of copper in solution. The presence of organic acids also can lead to the mobilization of copper from the sediments to solution. Copper has a strong affinity for hydrous iron and manganese oxides, clays, carbonate minerals, and organic matter. Sorption to these materials, both suspended in the water column and in the sediment, results in relative enrichment of the solid phase and reduction in dissolved levels. Sorption processes are quite efficient in scavenging dissolved copper and in controlling its mobility in natural unpolluted streams. The amounts of the various copper compounds and complexes that actually exist in solution depend on the pH, temperature, alkalinity, and concentrations of other chemical species. The levels of copper able to remain in solution are directly dependent on water chemistry. Generally, ionic copper is more soluble in low pH waters and less soluble in high pH waters.

As an essential nutrient, copper is accumulated by plants and animals, although apparently it is not generally biomagnified. Because copper is strongly bioaccumulated and because biogenic ligands play an important role in complexing copper, biological activity is a major factor in determining the distribution and occurrence of copper in the ecosystem. For example, bioaccumulation patterns may exhibit seasonal variations related to biological activity.

Because many copper compounds and complexes are readily soluble, copper is among the more mobile heavy metals in soil and other surface environments. The major process that limits the environmental mobility of copper is adsorption to organic matter, clays, and other materials. Atmospheric transport of copper compounds can also occur.

Health Effects

Copper appears to increase the mutagenic activity of triose reductone and ascorbic acid in bacterial test systems. However, copper itself does not appear to have mutagenic, teratogenic or carcinogenic effects in animals or humans. Dietary levels of trace elements such as molybdenum, sulfur, zinc, and iron can affect the level of copper that produces certain deficiency or toxicity symptoms. In general, more attention is given to the problems associated with copper deficiency than to problems of excess copper in the environment. However, high levels of copper can be toxic to humans.

Exposure to metallic copper dust can cause a short-term illness similar to metal fume fever that is characterized by chills, fever, aching muscles, dryness of mouth and throat, and headache. Exposure to copper fumes can produce upper respiratory tract irritation, a metallic or sweet taste, nausea, metal fume fever, and sometimes discoloration of skin and hair. Individuals exposed to dusts and mists of copper salts may exhibit congestion of nasal mucous membranes, sometimes of the pharynx, and occasionally ulceration with perforation of the nasal septum.

If sufficient concentrations of copper salts reach the gastrointestinal tract, they act as irritants and can produce salivation, nausea, vomiting, gastritis, and diarrhea. Elimination of ingested ionic copper by vomiting and diarrhea generally protects the patient from more serious systemic toxic effects, which can include hemolysis, hepatic necrosis, gastrointestinal bleeding, oliguria, azotemia, hemoglobinuria, hematuria, proteinuria, hypotension, tachycardia, convulsions, and death. Chronic exposure may result in anemia.

Copper salts act as skin irritants producing an itching eczema. Conjunctivitis or even ulceration and turbidity of the cornea may result from direct contact of ionic copper with the eye.

Toxicity to Wildlife and Domestic Animals

Mean acute toxicity values for a large number of freshwater animals range from 7.2 µg/liter for Daphnia pulicaria to 10,200 µg/liter for the bluegill. Toxicity tends to decrease as hardness, alkalinity, and total organic carbon increase. Chronic values for a variety of freshwater species range from 3.9 µg/liter for brook trout to 60.4 µg/liter for northern pike. Hardness does not appear to affect chronic toxicity. The acute-chronic ratios for different species range from 3 to 156. The more sensitive species tend to have lower ratios than the less sensitive species. In addition, the ratio seems to increase with hardness. Acute toxicity values for saltwater organisms range from 17 µg/liter for a calanoid copepod to 600 µg/liter for the shore crab. A chronic value of 54 µg/liter and an acute-chronic ratio of 3.4 is reported for the mysid shrimp. Long-term exposure to 5 µg/liter is fatal to the bay scallop.

Bioconcentration factors in freshwater species range from zero for the bluegill to 2,000 for the alga Chlorella regularis. Among saltwater species, the highest bioaccumulation factors are those for the bivalve molluscs. Oysters can bioaccumulate copper up to 28,200 times without any significant mortality.

Sheep are very susceptible to copper toxicosis, and poisoning may be acute or chronic. Acute poisoning is caused by direct action of copper salts on the gastrointestinal tract, resulting in gastroenteritis, shock, and death. The toxic dose is about 200 mg/kg and is usually obtained through an accidental overdose of an antihelminthic. Ingestion of excess copper over a long period of time results in absorption and accumulation of copper by the liver. This type of chronic cumulative poisoning may suddenly develop into an acute hemolytic crisis. Copper intake of 1.5 g/day for 30 days is known to be fatal for many breeds of sheep. Excessive copper may be stored in the liver as a result of excess copper ingestion, as a consequence of impaired liver function, or in connection with a deficiency or excess of other trace elements. Sheep eliminate accumulated copper very slowly after cessation of exposure.

Swine develop copper poisoning at levels of 250 mg/kg in the diet unless zinc and iron levels are increased. Toxicosis develops with hypochromic microcytic anemia, jaundice, and marked increases in liver and serum copper levels as well as serum aspartate amino transferase. High copper levels may be found in swine because of the practice of feeding them high copper diets in order to increase daily weight gain. However, swine rapidly eliminate copper once it is removed from the diet. Cattle are much more resistant to copper in the diet than sheep or swine. Copper toxicity in ruminants can be counteracted by including molybdenum and sulfate in the diet.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed)

Freshwater

Acute toxicity: $e^{(0.905\ [\ln(\text{hardness})] - 1.413)}$ µg/liter

Chronic toxicity: $e^{(0.905\ [\ln(\text{hardness})] - 1.785)}$ µg/lite

Saltwater

Acute toxicity: 3.2 µg/liter
Chronic toxicity: 2.0 µg/liter

Human Health

Organoleptic criterion: 1 mg/liter

National Secondary Drinking Water Standards (USEPA): 1 mg/liter

OSHA Standards: 1.0 mg/m^3 TWA (dust and mist)
0.1 mg/m^3 TWA (fume)

ACGIH Threshold Limit Values: 1.0 mg/m^3 TWA (dusts and mists)
0.2 mg/m^3 TWA (fume)
2.0 mg/m^3 STEL (dusts and mists)

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CRESOLS

Summary

Dermal application of cresols promotes skin tumors in mice. Cresols are highly irritating to the skin, mucous membranes, and eyes. They can impair liver and kidney function and cause central nervous system disturbances.

CAS Number: 1319-77-3

Chemical Formula: $CH_3C_6H_4OH$

IUPAC Name: *m*-Cresol, *o*-cresol, *p*-cresol

Important Synonyms and Trade Names: Cresylic acid, cresylol, tricresol

Chemical and Physical Properties

Molecular Weight: 108.13

Boiling Point: 191-203°C

Melting Point: 10.9-35.5°C

Specific Gravity: 1.030-1.038 at 25°C

Solubility in Water:
m-Cresol: 23,500 mg/liter at 20°C
o-Cresol: 31,000 mg/liter at 40°C
p-Cresol: 24,000 mg/liter at 40°C

Solubility in Organics: Miscible with alcohol, benzene, ether, and glycerol

Log Octanol/Water Partition Coefficient: 2 (calculated)

Vapor Pressure: 1 mm Hg at 38-53°C

Vapor Density: 3.72

pka: 10

Flash Point: 80°C

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Transport and Fate

From the available information, it appears that cresol is not very volatile and that the main transport process in the environment is movement in water. In aerated surface waters, it is photooxidized; and it may also be nonphotolytically oxidized by a metal-catalyzed reaction in some of these environments. In addition, biodegradation by water and soil microorganisms is apparently an important fate process. Because of the two competing fate processes, cresol probably is not very persistent in the environment.

Health Effects

None of the cresol isomers is regarded as a carcinogenic initiator. However, it has been reported that *o*-, *p*-, and *m*-cresol administered to mice as 20% solutions *in* benzene twice *weekly* for 20 weeks promoted papillomas initiated by a single dermal application of 9,10-dimethyl-1,2-benzanthracene (DMBA) (Boutwell and Bosch 1959). The mutagenicity and teratogenicity of the cresols have not been adequately assessed.

Cresols are highly irritating to the skin, mucous membranes, and eyes. Occupational exposure to cresols has caused severe burns and eczema. Although cresol isomers have relatively low vapor pressures, airborne cresols have reportedly caused headache, vomiting, and digestive disorders.

In addition to being strong irritants, cresols may impair kidney and liver functioning and cause central nervous system and cardiovascular disturbances. The rat oral LD_{50} values for *o*-, *p*-, and *m*-cresol are 135 mg/kg, 180 mg/kg, and 202 mg/kg, respectively. The dermal LD_{50} values for rabbits are 1,380 mg/kg and 2,050 mg/kg for the *o*- and *m*- isomers of cresol, respectively.

Toxicity to Wildlife and Domestic Animals

Waterborne cresol isomers are toxic to fish and other forms of aquatic life. Trout embryos are one of the most sensitive species, with 24-hour median threshold limits (TL_m) of 2 mg/liter for *o*-cresol, 7 mg/liter for *p*-cresol, and 4 mg/liter for *m*-cresol. The 24- to 96-hour TL_m for the bluegill is approximately 21.5 mg/liter for *o*-cresol and 11.8 mg/liter for the *p*- isomer. The LD_0 value for the alga *Scenedesmus* is 40 mg/liter for *o*- and *p*- isomers but 6 mg/liter for *m*-cresol. There is no evidence available that the cresols bioaccumulate in the tissues of wildlife species. No alterations in reproductive capabilities or other subtle changes in wildlife species have been attributed to these compounds.

Regulations and Standards

NIOSH Recommended Standard: 10 mg/m^3 TWA

OSHA Standards (skin): 20 mg/m^3

ACGIH Threshold Limit Value: 22 mg/m^3

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CYANIDE

Summary

Cyanide can be present in many forms in the environment. The transport, fate, and toxicity of the chemical is quite dependent on the specific form. Hydrogen cyanide and its simple salts are highly toxic following acute exposure by humans, experimental animals, and both aquatic and terrestrial wildlife.

Background Information

Cyanide (CN-) is usually defined as hydrogen cyanide (HCN) and its salts. The chemical/physical properties, transport and fate, and toxicity of cyanide are quite dependent on the form of cyanide present.

CAS Number: 151-50-8; 143-33-9

Chemical Formula: CN-

IUPAC Name: Cyanide

Chemical and Physical Properties

Molecular Weight: 27 (HCN)

Boiling Point: 26.7°C (HCN)

Melting Point: -14°C (HCN)

Specific Gravity: 0.699 at 22°C (HCN)

Solubility in Water: Soluble (HCN)

Solubility in Organics: Soluble in alcohol and ether

Vapor Pressure: 657.8 mm Hg at 21.9°C (HCN)

Transport and Fate

The transport and fate of cyanide in the environment is dependent on the chemical compound containing the cyanide. Most free cyanide will be HCN in aquatic environments and will probably evaporate, although biodegradation is another possible fate process. Metal cyanides are generally insoluble and for that reason will accumulate in the sediment. Sorption occurs but is not considered an important transport or fate process. Cyanides move rather freely in soils but biodegradation would probably significantly decrease the amount present in the groundwater. Volatilization of HCN and nitriles may occur from soil surfaces.

Health Effects

Hydrogen cyanide and its simple salts, such as sodium cyanide, are highly toxic by all routes. Many reports are available regarding acute poisoning in humans. Hydrogen cyanide vapor is irritating at very low concentrations, is considered dangerous at 20 ppm (20 mg/m^3), and is fatal at concentrations of 100 ppm (100 mg/m^3) for one hour. NIOSH notes reports of chronic poisoning resulting in fatigue, weariness and other subjective symptoms in workers, but these findings have been disputed by other investigators. Chronic exposure to low levels of cyanide salts has been reported to cause enlargement of the thyroid gland in humans, apparently due to inefficient elimination of the cyanide metabolite thiocyanate. NIOSH (1976) concluded that there was no evidence of carcinogenicity, mutagenicity, or teratogenicity for cyanides. Cyanide has been shown to produce chromosome breaks in a plant, Vicia faba. Because of its mechanism of action, inhibition of the election transport system in oxidative phosphorylation, cyanide is acutely toxic to almost all forms of life. A reduction in the TLV for HCN from 10 mg/m^3 to a ceiling value of 3 mg/m^3 has been recommended by several investigators, to prevent the various nonspecific effects noted by several investigators (ACGIH 1980).

Toxicity to Wildlife and Domestic Animals

Cyanide is acutely toxic to both freshwater and saltwater organisms, causing death at levels of about 50 µg/liter in sensitive species and being fatal to many species at levels above 200 µg/liter. Final acute values were determined to be 44.7 µg/liter for freshwater species and 2.03 µg/liter for saltwater species. Effects such as reduced survival and reduced reproduction were seen in fish chronically exposed to free cyanide concentrations of from 10 to 50 µg/liter. The final acute chronic ratios were determined to be 10.7 and 3.5 for freshwater and saltwater organisms, respectively. The final chronic values were determined by dividing the acute values by the acute-chronic ratio, and were determined to be 4.2 for freshwater species and 0.57 for saltwater organisms. An accidental spill of cyanide caused the death of 4,800 fish in Oak Ridge, Tennessee. The long-term effects of this spill were not reported. Livestock death and environmental damage were caused by high levels of cyanide leaching from a drum disposal site in Illinois.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed)

Freshwater

Acute toxicity: 22 µg/liter
Chronic toxicity: 4.2 µg/liter

Saltwater

Acute toxicity: 1.0 µg/liter
Chronic toxicity: 0.57 µg/liter

Human Health

Criterion: 200 µg/liter

Primary Drinking Water Standard (USEPA): 200 µg/liter

ACGIH Threshold Limit Value: 5 mg/m^3 TWA

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CYANURIC ACID

Summary

Cyanuric acid has been used as a selective herbicide. There is limited evidence that it may cause tumors in mice and rats. Chronic exposure to high doses can cause kidney damage.

CAS Number: 108-80-5

Chemical Formula: $C_3H_3N_3O_3$

IUPAC Name: Cyanuric acid

Important Synonyms and Trade Names: Isocyanuric acid; sym-triazinetriol; 1,3,5-triazine-2,4,6 (1H,3H,5H)-trione; 2,4,6-trihydroxy-1,3,5-triazine; tricyanic acid; trihydroxycyanidine

Chemical and Physical Properties

Molecular Weight: 129.08

Boiling Point: Decomposes

Melting Point: Higher than 360°C (decomposes)

Specific Gravity: 2.5 at 20°C

Solubility in Water: 2.5 to 5 g/liter

Solubility in Organics: Insoluble in cold methanol, ether, acetone, benzene, chloroform; soluble in hot alcohols

Transport and Fate

Cyanuric acid exists primarily in two equilibrating tautomeric species: a trioxo form and a trihydroxy form. The trioxo form is thought to predominate in the crystalline form of this compound and in solution. The trihydroxy form predominates in basic solution. Very little information concerning environmental transport and fate is available. In general, cyanuric acid is chemically stable and relatively inert. Volatilization and atmospheric transport are not likely to be significant environmental processes. Based on its low solubility in organic solvents, sorption to organic particulates and bioaccumulation probably do not occur to any significant extent. When administered to experimental animals at high doses, most of the compound is excreted unchanged. Some biodegradation has been reported to occur, and it may be the predominant fate process for cyanuric acid.

Health Effects

Cyanuric acid exhibited a low tumorigenic potential in one study with rats and mice exposed by subcutaneous, oral, and dermal routes. Tumors appeared after latent periods of more than 18 months. The study was conducted without concurrent controls, and the results must be considered equivocal. No mutagenic, teratogenic, or reproductive effects resulting from exposure to cyanuric acid have been reported. In general, cyanuric acid appears to have a low degree of toxicity. Chronic exposure at high doses has produced kidney changes. The oral LD_{50} value for the rat is 500 mg/kg.

Toxicity to Wildlife and Domestic Animals

Adequate data to characterize the toxicity to wildlife and domestic animals are not available.

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DDT

Summary

DDT is an organochlorine pesticide, which together with its metabolites, is very persistent in the environment. DDT, DDE, and DDD have been shown to be carcinogenic in mice. They primarily cause liver tumors, but they also increase the incidence of lung tumors and lymphomas. In addition, DDT is a reproductive toxin. Chronic exposure can damage the central nervous system and liver. DDT and other organochlorine pesticides are highly toxic to aquatic organisms and are responsible for the decreased reproductive success of many bird species.

Background Information

Technical DDT is a mixture containing 65-80% p,p'-DDT, 15-20% o,p'-DDT, up to 4% p,p'-DDD, and traces of other materials. Metabolites of DDT include p,p'-DDE and o,p'-DDD. The DDT isomers and metabolites are usually found together and generally have similar properties; therefore, they will be considered together. Where differences occur the specific isomer will be identified. DDT will be used to refer to the combination of technical material and metabolites. Specific DDT isomers will be identified as such.

CAS Number:	p,p'-DDT:	50-29-3
	o,p'-DDT:	789-02-6
	p,p'-DDD:	72-54-8
	o,p'-DDD:	53-19-0
	p,p'-DDE:	72-55-9

Chemical Formula:	p,p'- and o,p'-DDT:	$C_{14}H_9Cl_5$
	p,p'- and o,p'-DDD:	$C_{13}H_{10}Cl_4$
	p,p'- and o,p'-DDE:	$C_{14}H_8Cl_4$

IUPAC Name:	p,p'-DDT:	1,1,1-Trichloro-2,2-bis(4-chlorophenyl) ethane
	o,p'-DDT:	1,1,1-Trichloro-2-(2-chlorophenyl)-2-(4-chlorophenyl)ethane
	p,p'-DDD:	1,1-Dichloro-2,2-bis(4-chlorophenyl)-ethane
	o,p'-DDE:	1,1-Dichloro-2,2-bis(4-chlorophenyl)-ethene

Important Synonyms and Trade Names:

DDT: Dichlorodiphenyltrichloroethane, dicophane, chlorophenothane, Gesarol, Neocid
p,p'-DDD: TDE, Rothane

Chemical and Physical Properties

Molecular Weight: o,p'- and p,p'-DDT: 354.5
DDD: 320
DDE: 318

Boiling Point: DDT: 260°C

Melting Point: DDT: 109°C
DDD: 112°C
DDE: 90°C

Solubility in Water: p,p'-DDT: 5.5 μg/liter
o,p'-DDT: 26 μg/liter
p,p'-DDD: 20 μg/liter
DDE: 14 μg/liter

Solubility in Organics: DDT: Soluble in acetone, benzene, cyclohexanane, morpholine, pyridine, and dioxane

Log Octanol/Water Partition Coefficient:

DDT: 4.98
p,p'-DDT: 3.98
p,p'-DDD: 5.99
o,p'-DDD: 6.08
DDE: 5.69

Vapor Pressure:

p,p'-DDT: $1.9x10^{-7}$ mm Hg at 25°C
p,p'-DDT: $7.3x10^{-7}$ mm Hg at 30°C
o,p'-DDT: $5.5x10^{-6}$ mm Hg at 30°C
p,p'-DDD: $1.0x10^{-6}$ mm Hg at 30°C
o,p'-DDD: $1.9x10^{-6}$ mm Hg at 30°C
DDE: $6.5x10^{-6}$ mm Hg at 20°C

Transport and Fate

DDT and its metabolites are very persistent in the environment. Volatilization is probably the most important transport process from soil and water for p,p'-DDT and o,p'-DDT, as evidenced by the ubiquitous nature of DDT in the environment.

Sorption and bioaccumulation are the most important transport processes for the DDT isomers. Although it only occurs slowly, the ultimate fate process for p,p'-DDT, o,p'-DDT, and DDD is biotransformation to form bis(2-chlorophenyl)methanone (DDCO). Indirect photolysis may also be important for p,p'-DDT and o,p'-DDT in aquatic environments. For DDE, direct photolysis is the most important ultimate fate process in the environment, although biotransformation may also be important.

Health Effects

DDT, DDE, and DDD have been shown to be carcinogenic to mice, primarily causing liver tumors, but also causing lung tumors and lymphomas. DDT does not appear to be mutagenic, but it has caused chromosomal damage. There is no evidence that DDT is a teratogen; but it is a reproductive toxin, causing reduced fertility, reduced growth of offspring, and fetal mortality.

Chronic exposure to DDT causes a number of adverse effects, especially to the liver and central nervous system (CNS). DDT induces various microsomal enzymes and therefore probably affects the metabolism of steroid hormones and exogenous chemicals. Other effects on the liver include hypertrophy of the parenchymal cells and increased fat deposition. In the CNS, exposure to DDT causes behavioral effects such as decreased aggression and decreased conditional reflexes. Acute exposure to large doses or chronic exposure to lower doses causes seizures. The oral LD_{50} is between 113 and 450 mg/kg for the rat and is generally higher for other animals.

DDT, DDD, and DDE are bioconcentrated and stored in the adipose tissues of most animals.

Toxicity to Wildlife and Domestic Animals

DDT has been extensively studied in freshwater invertebrates and fishes and is quite toxic to most species. The range of toxicities was 0.18 to 1,800 µg/liter and the freshwater final acute value for DDT and its isomers was determined by EPA to be 1.1 µg/liter. Saltwater species were somewhat more sensitive to DDT; the saltwater final acute value for the DDT isomers was 0.13 µg/liter. Only one chronic toxicity test on aquatic species was reported. This test indicated that the acute-chronic ratio for DDT might be high (65 in the reported study), but the data were insufficient to allow calculation of a final acute-chronic ratio. DDT, DDD, and DDE are bioconcentrated by a factor of 10^3 to 10^5.

DDT, DDD, DDE and the other persistent organochlorine pesticides are primarily responsible for the great decrease in the reproductive capabilities and consequently in the populations of fish-eating birds, such as the bald eagle, brown pelican, and osprey. DDT has also been shown to decrease the populations of numerous other species of waterbirds, raptors, and passerines significantly.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

DDT: Freshwater

Acute toxicity: 1.1 μg/liter
Chronic toxicity: 0.001 μg/liter

Saltwater

Acute toxicity: 0.13 μg/liter
Chronic toxicity: 0.001 μg/liter

DDD and DDE: The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: DDD: 0.6 μg/liter
DDE: 1050 μg/liter
Chronic toxicity: DDD & DDE: No available data

Saltwater

Acute toxicity: DDD: 3.6 μg/liter
DDE: 14 μg/liter
Chronic toxicity: DDD & DDE: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of DDT in water are:

Risk	Concentration
10^{-5}	0.24 ng/liter
10^{-6}	0.024 ng/liter
10^{-7}	0.0024 ng/liter

CAG Unit Risk (USEPA): 0.34 $(mg/kg/day)^{-1}$

OSHA Standard (air): 1 mg/m^3 TWA

ACGIH Threshold Limit Value: 1 mg/m^3 TWA

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DIBROMOCHLOROPROPANE

Summary

Dibromochloropropane (DBCP) was formerly used as a soil fumigant and nematocide. It has been found to be carcinogenic in mice and rats. It causes mammary tumors (in female rats only) and forestomach tumors when administered orally, and nasal, tongue, and lung tumors when given by inhalation. Men occupationally exposed to DBCP had abnormally low sperm counts. Animals studies have shown that dibromochloropropane has adverse effects on the liver, kidneys, and blood cells.

CAS Number: 96-12-8

Chemical Formula: $C_3H_5Br_2Cl$

IUPAC Name: 1,2-Dibromo-3-chloropropane

Important Synonyms and Trade Names: DBCP, Fumazone, Nemagon

Chemical and Physical Properties

Molecular Weight: 236.36

Boiling Point: 196°C

Melting Point: 6°C

Specific Gravity: 2.093 at 14°C

Solubility in Water: Slightly soluble (probably 5-10 g/liter)

Solubility in Organics: Miscible with oils, dichloropropane, and isopropyl alcohol

Vapor Pressure: 0.8 mm Hg at 21°C

Transport and Fate

There was no information available on the transport and fate of 1,2-dibromo-3-chloropropane (DBCP) at the time of this review. However, there is some information on the transport and fate of structurally similar compounds that may be relevant to the environmental fate of DBCP.

1,2,3-Trichloropropane was found to have a half-life of 51 minutes in stirred water, suggesting volatilization of DBCP from water could be significant. However, DBCP is considerably heavier than 1,2,3-trichloropropane and thus somewhat less likely to volatilize. The log octanol/water partition coefficient, 2.28, of 1,2-dichloropropane suggests that it will readily adsorb to organic components of soils and sediments and, therefore, be transported in dust and suspended solids. The tendency of brominated aliphatics to have higher log octanol/water portion coefficients than chlorinated aliphatics suggest DBCP will adsorb to a greater degree than 1,2-dichloropropane. Because of its water solubility, density, and low vapor pressure, DBCP is a likely groundwater contaminant. Its high density suggests that it would settle to the bottom of a contaminant plume and ultimately to the bottom of the aquifer.

Based on information of one and two carbon aliphatics, DBCP may be oxidized in the troposphere by hydroxyl radicals and hydrolyzed in an aqueous environment. Biodegradation of 1,2-dichloropropane does occur by soil microorganisms. However, the amount and speed of biodegradation and chemical degradation of DBCP is unknown.

Health Effects

DBCP has been found to be carcinogenic in two animal bioassays and mutagenic in the Ames assay system. In a gavage study, DBCP was found to produce significantly increased incidences of squamous-cell carcinomas of the forestomach of mice and rats and of mammary adenocarcinomas in female rats. In an inhalation study, rats had increased incidences of nasal cavity tumors and tumors of the tongue, while mice had increased incidences of nasal cavity tumors and lung tumors.

Men occupationally exposed to DBCP during its manufacture were found to have abnormally low sperm counts. Male rats exposed to DBCP during subchronic toxicity studies were also found to have abnormally low sperm cells as well as degenerative changes in the seminiferous tubules, decreased weight of the testes, and an increased proportion of abnormal sperm cells. Liver and kidney effects have also been noted in animal studies. Effects range from dilatation of the sinusoids and centrilobular congestion to cirrhosis and necrosis in the liver. Cloudy swelling of the epithelium of the proximal convoluted tubules and increased amounts of interstitial tissue have been found in the kidneys. Effects on blood cells were also noted in several studies. These effects include severe leukopenias and anemias in exposed monkeys and decreased activity of phagocytic cells in exposed rats.

Toxicity to Wildlife and Domestic Animals

The oral LD_{50} value of DBCP to young mallards is 66.5 mg/kg, which is lower than the oral LD_{50} value for the rat and mouse--173 and 257 mg/kg, respectively. Exposure to a water concentration of 1 mg/liter DBCP for 24 hours produced a 90% mortality in clam larve. At a use concentration of 20 gallons DBCP per acre, 100% of exposed earthworms died in 1 day. At a use rate of 5 pounds per acre, DBCP killed 87% of the Lumbricus and 28% of the Helodrilus sp. in 32 days.

Standards and Regulations

NIOSH Recommended Standard: 10 ppb (0.1 mg/m^3)

OSHA Standard (air): 1 ppb (9.6 $\mu g/m^3$) TWA

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Summary

Dichlorobenzene (DCB) is probably persistent in the natural environment. In rats, chronic oral exposure to dichlorobenzene caused liver and kidney damage and changes in the hematopoietic system. In humans, DCB is a skin and eye irritant; inhalation exposure causes nausea and irritates the membranes.

CAS Number: 1,2-Dichlorobenzene (1,2-DCB) 95-50-1
1,3-Dichlorobenzene (1,3-DCB) 541-73-1
1,4-Dichlorobenzene (1,4-DCB) 106-46-7

Chemical Formula: $C_6H_4Cl_2$

IUPAC Name: Dichlorobenzene

Important Synonyms and Trade Names: Dichlorobenzene, DCB

Chemical and Physical Properties

Molecular Weight: 147.01

Boiling Point: 1,2-DCB: 180.5°C
1,3-DCB and 1,4-DCB: 173°C

Melting Point: 1,2-DCB:-17.0°C
1,3-DCB:-24°C
1,4-DCB:-53°C

Specific Gravity: 1.3 at 20°C

Solubility in Water: 1,2-DCB: 145 mg/liter at 25°C
1,3-DCB: 123 mg/liter at 25°C
1,4-DCB: 80 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, ether, acetone, benzene, carbon tetrachloride, and ligroin

Log Octanol/Water Partition Coefficient: 3.38

Vapor Pressure: 1 mm Hg at 20°C

Vapor Density: 5.05

Henry's Law Constant: 1.99×10^{-3} atm m^3/mole

Flash Point: 71°C

Transport and Fate

Relatively little information concerning the environmental fate of dichlorobenzene (DCB) is available. DCB is expected to volatilize at a relatively rapid rate, and atmospheric transport can occur. It has an estimated half-life for removal from agitated surface water of 9 hours or less. Dichlorobenzenes are reported to be reactive toward hydroxyl radicals in air with a half-life of about 3 days, but indirect evidence suggests that DCB does not hydrolyze at a significant rate under normal environmental conditions. The high log octanol/water partition coefficient for DCB suggests that adsorption to organic matter in aquatic systems and soil is probably an important environmental fate process. Indirect evidence suggests that bioaccumulation may also be an important fate process. DCB appears to be resistant to biodegradation. However, it may be broken down to some degree by pollutant-acclimatized microorganisms. Sorption, bioaccumulation, and volatilization with subsequent atmospheric oxidation are likely to be competing processes, with the dominant fate being determined by local environmental conditions. If volatilization doesn't occur, dichlorobenzene is probably rather persistent.

Health Effects

It is generally thought that the available data are inadequate for assessing the carcinogenic potential of DCB in animals and humans. One case study suggests an association between exposure to dichlorobenzene and several cases of leukemia. DCB is reported to be nonmutagenic in Salmonella typhimurium tester strains. Mutagenic and clastogenic activity reportedly occurs in some plant test systems. No data are available for evaluating the teratogenic or reproductive effects in animals or humans.

Symptoms of acute inhalation intoxication in humans include headache, nausea, and throat irritation. DCB is also a skin and eye irritant.

A variety of other symptoms, including weakness, fatigue, and anemia, have been observed after chronic dermal and inhalation exposure to dichlorobenzene.

Inhalation of DCB causes eye and upper respiratory tract irritation, central nervous system depression, and liver and kidney damage in experimental animals. An LC_{20} of approximately

4,900 mg/m^3/7 hours is reported for the rat. No toxic effects were observed after daily 7-hour inhalation exposures of up to 560 mg/m^3 for as much as 7 months in several species of experimental animals. Hepatic porphyria is reported to occur in rats after daily tracheal intubation of 455 mg/m^3 for up to 15 days. Oral exposure results in stimulation of liver microsomal enzyme systems and cumulative toxicity. The oral LD_{50} for the rat is 500 mg/kg. Chronic oral exposure to 188 mg/kg/day causes liver and kidney damage in rats. Exposure to 0.01-0.1 mg/kg/day produces changes in the hematopoietic system, increased prothrombin time, and altered conditioned reflexes and enzyme activities in chronically exposed rats. In general, toxicity increases in the order 1,4-DCB, 1,3-DCB, 1,2-DCB.

Toxicity to Wildlife and Domestic Animals

The 48-hour and 96-hour LC_{50} values for Daphnia and bluegills, respectively, tested under static conditions, were 2,440 and 5,590 µg/liter (1,2-DCB); 28,100 and 5,020 µg/liter (1,3-DCB); and 11,000 and 4,280 µg/liter (1,4-DCB). Two flow through 96-hour LC_{50} tests using fathead minnows and rainbow trout gave values of about 3,000 µg/liter. A freshwater chronic value of 2,000 µg/liter is reported for the fathead minnow. Acute values for three saltwater species ranged from 1,970 µg/liter for the mysid shrimp to 9,660 µg/liter for the sheepshead minnow. No saltwater chronic values are available. A whole body bioconcentration factor of about 80 is reported for the bluegill.

The 96-hour median effect levels for chlorophyll a and cell number are 179,000 and 149,000 µg/liter, respectively, in the freshwater alga *Selenastrum capricornutum*. In the saltwater alga *Skeletonema costatum* the corresponding values are 44,200 and 44,100 µg/liter, respectively.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 400 µg/liter

OSHA Standard: 300 mg/m^3 Ceiling Level

ACGIH Threshold Limit Value: 300 mg/m^3 Ceiling Level

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1,1-DICHLOROETHANE

Summary

1,1-Dichloroethane is quite volatile and probably is not very persistent in aquatic environments. Inhalation exposure to high doses causes central nervous system depression in humans and may cause hepatotoxicity. In animals, high doses cause liver and kidney damage and retard fetal development.

CAS Number: 75-34-3

Chemical Formula: CH_3CHCl_2

IUPAC Name: 1,1-Dichloroethane

Important Synonyms and Trade Names: Ethylidene chloride, ethylidene dichloride

Chemical and Physical Properties

Molecular Weight: 98.96

Boiling Point: 57.3°C

Melting Point: -97.0°C

Specific Gravity: 1.1776 at 20°C

Solubility in Water: 5 g/liter

Solubility in Organics: Miscible in alcohol

Log Octanol/Water Partition Coefficient: 1.79

Vapor Pressure: 180 mm Hg at 20°C

Transport and Fate

1,1-Dichloroethane disperses from surface water primarily by volatilization into the troposphere, where it is subsequently broken down by hydroxylation. No studies on adsorption were found in the literature reviewed, but because of its water solubility and relatively low log octanol/water partition coefficient, 1,1-dichloroethane potentially could move through soil and enter the groundwater.

Health Effects

Limited toxicological testing of 1,1-dichloroethane has been conducted, although the literature indicates that 1,1-dichloroethane is one of the least toxic of the chlorinated ethanes. An NCI bioassay on 1,1-dichloroethane was limited by poor survival of test animals of test animals, but some marginal tumorigenic effects were seen. Inhalation exposure to high doses of 1,1-dichloroethane (over 16,000 mg/m^3) caused retarded fetal development in rats (Schwetz et al. 1974). 1,1-Dichloroethane was not found to be mutagenic using the Ames assay. 1,1-Dichloroethane causes central nervous system depression when inhaled at high concentrations, and evidence suggests that the compound is hepatotoxic in humans. Kidney and liver damage was seen in animals exposed to high levels of 1,1-dichloroethane. The oral LD$_{50}$ value in the rat is 725 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of 1,1-dichloroethane to aquatic species was reported in the literature reviewed. However, the available information on the chloroethanes indicates that toxicity declines with decreases in chlorination and that the 1,1,1-isomer is less active than the 1,1,2-isomer. Therefore 1,1-dichloroethane is probably no more toxic than 1,2-dichloroethane, which is acutely toxic at levels of 100-500 mg/liter and has a chronic toxicity beginning at about 20 mg/liter.

No information on the toxicity of 1,1-dichloroethane to terrestrial wildlife or domestic animals was found in the sources reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data were inadequate for establishing criteria.

OSHA Standard (air): 400 mg/m^3 TWA

ACGIH Threshold Limit Value: 810 mg/m^3 TWA

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1,2-DICHLOROETHANE

Summary

1,2-Dichloroethane (ethylene dichloride) is a volatile organic solvent, and volatilization and percolation into groundwater may be significant routes of transport. It has a low solubility in water and may be a component in nonaqueous-phase liquids. 1,2-Dichloroethane is carcinogenic in animals and mutagenic in bacterial test systems; it is a suspected human carcinogen.

CAS Number: 107-06-2

Chemical Formula: CH_2ClCH_2Cl

IUPAC Name: 1,2-Dichloroethane

Important Synonyms and Trade Names: Ethylene dichloride, glycol dichloride

Chemical and Physical Properties

Molecular Weight: 98.96

Boiling Point: 83-84°C

Melting Point: -35.4°C

Specific Gravity: 1.253 at 20°C

Solubility in Water: 8 g/liter

Solubility in Organics: Miscible with alcohol, chloroform, and ether

Log Octanol/Water Partition Coefficient: 1.48

Vapor Pressure: 61 mm Hg at 20°C

Flash Point: 15°C (closed cup)

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Transport and Fate

The primary method of dispersion from surface water for 1,2-dichloroethane is volatilization. In the atmosphere, 1,2-dichloroethane is rapidly broken down by hydroxylation, although some may be absorbed by atmospheric water and return to the earth by precipitation. No studies on the adsorption of 1,2-dichloroethane onto soil were reported in the literature examined. However, 1,2-dichloroethane has a low octanol/water partition coefficient, is slightly soluble in water, and therefore leaching through the soil into the groundwater is an expected route of dispersal.

Health Effects

1,2-Dichloroethane is carcinogenic in rats and mice, producing a variety of tumors. When administered by gavage, it produced carcinomas of the forestomach and hemangiosarcomas of the circulatory system in male rats; adenocarcinomas of the mammary gland in female rats; lung adenomas in male mice; and lung adenomas, mammary adenocarcinomas, and endometrial tumors in female mice. It is mutagenic when tested using bacterial test systems. Human exposure by inhalation to 1,2-dichloroethane has been shown to cause headache, dizziness, nausea, vomiting, abdominal pain, irritation of the mucous membranes, and liver and kidney dysfunction. Dermatitis may be produced by skin contact. In severe cases, leukocytosis (an excess of white blood cells) may be diagnosed; and internal hemorrhaging and pulmonary edema leading to death may occur. Similar effects are produced in experimental animals.

Toxicity to Wildlife and Domestic Animals

1,2-Dichloroethane is one of the chlorinated ethanes least toxic to aquatic life. For both fresh- and saltwater species, it is acutely toxic at concentrations greater than 118 mg/liter, while chronic toxicity has been observed at 20 mg/liter. 1,2-Dichloroethane is not likely to bioconcentrate, as its steady state bioconcentration factor was 2 and its elimination half-life was less than 2 days in bluegill.

No information on the toxicity of 1,2-dichloroethane to domestic animals or terrestrial wildlife was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 118 mg/liter
Chronic toxicity: 20 mg/liter

Saltwater

Acute toxicity: 113 mg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of 1,2-dichloroethane in water are:

Risk	Concentration
10^{-5}	9.4 μg/liter
10^{-6}	0.94 μg/liter
10^{-7}	0.094 μg/liter

CAG Unit Risk (USEPA): 9.1×10^{-2} $(mg/kg/day)^{-1}$

OSHA Standards: 200 mg/m^3 TWA
400 mg/m^3 Ceiling Level
800 mg/m^3 for 5 min every 3 hr, Peak Concentration

ACGIH Threshold Limit Values: 40 mg/m^3 TWA
60 mg/m^3 STEL

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1,1-DICHLOROETHYLENE

Summary

1,1-Dichloroethylene (VDC, vinylidene chloride) caused kidney tumors (in males only) and leukemia in one study of mice exposed by inhalation, but the results of other studies were equivocal or negative. 1,1-Dichloroethylene is mutagenic, and it caused adverse reproductive effects when administered to rats and rabbits by inhalation. Chronic exposure causes liver damage, and acute exposure to high doses produces nervous system damage.

CAS Number: 75-35-4

Chemical Formula: CH_2CCl_2

IUPAC Name: 1,1-Dichloroethene

Important Synonyms and Trade Names: Vinylidene chloride, VDC, 1,1-dichloroethene, 1,1-DCE

Chemical and Physical Properties

Atomic Weight: 96.94

Boiling Point: 37°C

Melting Point: -122.1°C

Specific Gravity: 1.218 at 20°C

Solubility in Water: 400 mg/liter at 20°C

Solubility in Organics: Sparingly soluble in alcohol, ether, acetone, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 1.48

Vapor Pressure: 500 mm Hg at 20°C

Vapor Density: 3.25

Transport and Fate

Volatilization appears to be the primary transport process for 1,1-dichloroethylene (VDC), and its subsequent photooxidation in the atmosphere by reaction with hydroxyl radicals is apparently the predominant fate process. Information on other transport and fate mechanisms was generally lacking for 1,1-dichloroethylene. However, by inference from related compounds, hydrolysis, sorption, bioaccumulation, biotransformation, and biodegradation probably all occur but at rates too slow to be of much significance.

Health Effects

1,1-Dichloroethylene caused kidney tumors in males and leukemia in males and females in one study of mice exposed by inhalation, gave equivocal results in other inhalation studies, and gave negative results in rats and mice following oral exposure and in hamsters following inhalation exposure. VDC was mutagenic in several bacterial assays. 1,1-Dichloroethylene did not appear to be teratogenic but did cause embryotoxicity and fetotoxicity when administered to rats and rabbits by inhalation. Chronic exposure to oral doses of VDC as low as 5 mg/kg/day caused liver changes in rats. Acute exposure to high doses causes central nervous system depression, but neurotoxicity has not been associated with low-level chronic exposure. The oral LD_{50} value for the rat is 1,500 mg/kg, and for the mouse it is 200 mg/kg.

Toxicity to Wildlife and Domestic Animals

1,1-Dichloroethylene is not very toxic to freshwater or saltwater species, with acute LC_{50} values generally ranging from 80 to 200 mg/liter. A chronic study in which no adverse effects were observed indicated that the acute-chronic ratio was less than 40; a 13-day study that produced an LC_{50} of 29 mg/liter indicated that the acute-chronic ratio is greater than 4.

No reports of the toxicity of 1,1-dichloroethylene to terrestrial wildlife or domestic animals were found in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are inadequate for establishing criteria. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 11,600 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 224,000 μg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of 1,1-dichloroethylene in water are:

Risk	Concentration
10^{-5}	0.33 μg/liter
10^{-6}	0.033 μg/liter
10^{-7}	0.0033 μg/liter

CAG Unit Risk (USEPA): 1.16 $(mg/kg/day)^{-1}$

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1,2-trans-DICHLOROETHYLENE

Summary

Chronic inhalation exposure to 1,2-trans-dichloroethylene (1,2-trans-DCE) causes liver degeneration, and acute exposure to high levels has adverse effects on the central nervous system.

CAS Number: 540-59-0

Chemical Formula: $C_2H_2Cl_2$

IUPAC Name: 1,2-trans-Dichloroethene

Important Synonyms and Trade Names: trans-Acetylene dichloride, dioform

Chemical and Physical Properties

Molecular Weight: 96.94

Boiling Point: 47.5°C

Melting Point: -50°C

Specific Gravity: 1.2565 at 20°C

Solubility in Water: 600 mg/liter

Solubility in Organics: Miscible with alcohol, ether, and acetone; very soluble in benzene and chloroform

Log Octanol/Water Partition Coefficient: 1.48 (calculated)

Vapor Pressure: 200 mm Hg at 14°C

Flash Point: 3°C (undefined isomers)

Transport and Fate

Due to the relatively high vapor pressure of 1,2-trans-dichloroethylene (1,2-trans-DCE), volatilization from aquatic systems to the atmosphere is quite rapid and appears to be the primary transport process. Aerial transport of this compound can occur and is partly responsible for its relatively wide environmental distribution. Although little applicable information is available, adsorption is probably an insignificant environmental fate process for 1,2-trans-DCE. The relatively low log octanol/water partition coefficient of 1,2-trans-DCE suggests that bioaccumulation also is a relatively insignificant process. Although no information pertaining specifically to biodegradation of 1,2-trans-DCE is available, results with similar compounds suggest that this process probably occurs but at a very slow rate.

Photooxidation in the troposphere appears to be the dominant environmental fate of 1,2-trans-DCE. Once in the troposphere, the compound is attacked at the double bond by hydroxyl radicals, resulting in the formation of formic acid, hydrochloric acid, carbon monoxide, and formaldehyde. The half-life of 1,2-trans-DCE in the troposphere is estimated to be less than one day. Given the properties of similar compounds, photolysis of 1,2-trans-DCE in aquatic systems and photodissociation in the terrestrial environment are probably insignificant.

Health Effects

Very little information concerning exposure only to 1,2-trans-DCE is available. There are no reports of carcinogenic or teratogenic activity by 1,2-trans-DCE in animals or humans. It is reportedly nonmutagenic in a variety of test systems. Like other members of the chlorinated ethylene series, 1,2-trans-DCE has anesthetic properties. Exposure to high vapor concentrations has been found to cause nausea, vomiting, weakness, tremor, and cramps in humans. Repeated exposure via inhalation of 800 mg/m^3 (8 hours/day, 5 days/week, for 16 weeks) was reported to produce fatty degeneration of the liver in rats. The intraperitoneal injection LD_{50} value for the rat is 7,536 mg/kg.

Although nephrotoxic and cardiac sensitizing effects are associated with exposure to 1,1-dichloroethylene, the 1,2-DCE isomers have not been investigated with respect to this type of effects. 1,2-trans-Dichloroethylene can inhibit aminopyrine demethylation in rat liver microsomes in vitro, and it may thus interact with the hepatic drug-metabolizing monooxygenase system.

Toxicity to Wildlife and Domestic Animals

Practically no information concerning the toxicity of 1,2-trans-DCE to wildlife and domestic animals exists. The reported 96-hour LC_{50} value under static conditions is 135,000 μg/liter for the bluegill. Under the same test conditions, the LC_{50} value for 1,1-dichloroethylene is 73,900 μg/liter. Recommended criteria for protection of aquatic life are based primarily on data concerning 1,1-dichloroethylene.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data are not adequate for establishing criteria.

OSHA Standard: 790 mg/m^3 TWA

ACGIH Threshold Limit Values: 790 mg/m^3 TWA
1,000 mg/m^3 STEL

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2,4-DICHLOROPHENOL

Summary

2,4-Dichlorophenol (2,4-DCP) is not very persistent in the environment. There is equivocal evidence suggesting that it may act as a tumor promoter. Subcutaneous administration of 2,4-dichlorophenol to pregnant mice induced minor teratogenic effects. Chronic exposure caused nonspecific liver changes in mice.

CAS Number: 120-83-2

Chemical Formula: $C_6H_3Cl_2OH$

IUPAC Name: 2,4-Dichlorophenol

Important Synonyms and Trade Names: 2,4-DCP

Chemical and Physical Properties

Molecular Weight: 163.0

Boiling Point: 210°C

Melting Point: 45°C

Specific Gravity: 1.383 at 25°C

Solubility in Water: 4,500 mg/liter

Solubility in Organics: Soluble in benzene, alcohol, ether, and chloroform

Log Octanol/Water Partition Coefficient: 2.75

Vapor Pressure: 0.12 mm Hg at 20°C (calculated)

Vapor Density: 5.62

pKa: 7.48

Flash Point: 114°C

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Transport and Fate

2,4-Dichlorophenol (2,4-DCP) is not very persistent in the environment, with a half-life of about 1 week. Degradation by soil and water microorganisms occurs readily and appears to be the primary fate of 2,4-DCP. However, biodegradation rates are dependent on a number of environmental factors. For example, degradation will proceed much more quickly in systems containing pollutant-adapted microflora. Volatilization and adsorption do not appear to be significant transport processes for 2,4-dichlorophenol. Oxidation and hydrolysis are probably not important environmental fates. Photodegradation of aqueous 2,4-DCP is reported to occur under aerobic conditions, but it is unlikely that this process contributes significantly to its environmental fate. The limited data available suggest that 2,4-DCP does not readily bioaccumulate.

Health Effects

No studies evaluating the carcinogenic potential of 2,4-DCP are available. However, one study provides evidence that this compound may have promoting activity. Mice were initiated with skin applications of dimethyl-benzanthracene in benzene, and then received skin applications of 40.8 mg/kg of 2,4-DCP two times per week, for 15 or 24 weeks. The results indicated that 2,4-DCP can act as a promoter in the production of papillomas. However, the results must be regarded as equivocal because of limitations imposed by the experimental methods used in the study.

2,4-DCP is reported to have some effects on mitosis and meiosis in flower buds and root cells of Vicia faba. No studies evaluating the mutagenic activity of DCP in other eukayotic organisms or bacteria are available. In mice, subcutaneous administration of 74 mg/kg 2,4-DCP on days 6-14 of gestation resulted in a significant increase in abnormal fetuses, with half of the anomalies consisting of extended legs. Fetal mortality was unchanged, but weights were significantly lower than controls. No other reports of significant teratogenic or reproductive effects are available.

Very little information concerning the acute or chronic toxicity of 2,4-DCP is available. Acute toxicity following injection is characterized by initial polypnea followed by slowed respiration and dyspnea, hypotonia, coma, and death. A maximum no-effect level of 100 mg/kg/day was determined in a 6-month feeding study in mice. Only non-specific microscopic liver changes were observed in mice receiving 230 mg/kg/day. Intraperitoneal and oral LD_{50} values of 430 mg/kg and 580 mg/kg, respectively, are reported for the rat.

Toxicity to Wildlife and Domestic Animals

Species mean acute values reported for the freshwater species *Daphnia magna*, fathead minnow, and bluegill are 2,605, 8,230, and 2,020 μg/liter, respectively. A chronic value of 365 μg/liter and an acute-chronic ratio of 23 are reported for the fathead minnow. The only information available concerning saltwater species indicates that the mountain bass *Kuhlia sandvicensis* exhibits a moderate reaction in response to 20 mg/liter 2,4-DCP. Complete destruction of chlorophyll and 56.4% reduction of photosynthetic oxygen production are observed after exposure of the freshwater alga *Chlorella pyrenoidosa* to 100 and 50 mg/liter, respectively. The weighted average bioconcentration factor for 2,4-DCP and the edible portion of all freshwater and estuarine organisms consumed by Americans is calculated to be 40.7.

2,4-DCP residues have been detected in the liver and kidneys of cattle and chickens, and in chicken eggs. Concentrations of 2,4-DCP in animal tissues are reported to diminish rapidly after withdrawal of the 2,4-DCP precursor, 2,4-dichlorophenoxyacetic acid (2,4-D). No information concerning toxicity of 2,4-DCP to domestic animals is available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Health criterion: 3.09 mg/liter
Organoleptic criterion: 0.3 μg/liter

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2,4-DICHLOROPHENOXYACETIC ACID

Summary

2,4-Dichlorophenoxyacetic acid (2,4-D) is a commonly used broad spectrum herbicide. It is a component of Agent Orange, the defoliant most widely used in Vietnam. It promoted tumors after being painted on the skin of mice, and it probably is a weak mutagen. 2,4-D caused developmental abnormalities and was fetotoxic when administered to pregnant rats, mice, and hamsters. Dermal exposure to 2,4-D causes severe peripheral neuropathy.

CAS Number: 94-75-7

Chemical Formula: $Cl_2C_6H_3OCH_2COOH$

IUPAC Name: 2,4-Dichlorophenoxyacetic acid

Important Synonyms and Trade Names: Agrotect, Dicotox, Phenox, 2,4-D

Chemical and Physical Properties

Molecular Weight: 221.04

Boiling Point: 160°C at 0.4 mm Hg

Melting Point: 138°C

Solubility in Water: 620 mg/liter

Solubility in Organics: Soluble in organic solvents

Log Octanol/Water Partition Coefficient: 2.5 (calculated)

Vapor Pressure: $<10^{-5}$ mm Hg at 25°C

Vapor Density: 7.63

pKa: 2.8

Transport and Fate

Because of its low vapor pressure and relatively high solubility in water, 2,4-dichlorophenoxyacetic acid (2,4-D) is probably not very volatile. In surface water, 2,4-D undergoes either photolysis with oxidation to chlorophenols or photorewhich process occurs depends on the physical properties of the media. 2,4-D is only weakly adsorbed to soil and may leach into groundwater, although studies indicate that this is not an important transport process. Biodegradation by soil bacteria may be an important fate process for 2,4-D.

Health Effects

2,4-Dichlorophenoxyacetic acid has been assayed for carcinogenicity in rats, mice, and dogs. Statistically significant increases in tumor initiation have not been observed in any study. Increases in the number of lymphosarcomas, total sarcomas, and carcinomas in rats, however, suggest that it may be carcinogenic. A tumor-promoting effect was observed in a skin-painting study in mice.

2,4-D has damaged DNA and inhibited DNA repair in several strains of bacteria and yeast. It caused chromosomal damage and induced increased rates of sister chromatid exchange (SCE) in cultured human lymphocytes. 2,4-D also induced SCE in Chinese hamster ovary cells. The results of the Drosophila sex-linked recessive lethal assay were weakly positive. 2,4-D failed to induce mutation in the Ames assay. Considering all available test data, 2,4-D is a weak mutagen.

When administered to pregnant rats, mice, and hamsters, 2,4-D produces a pattern of developmental abnormalities, including skeletal anomalies and cleft palate. Fetotoxicity and fetal death have also been reported. The minimum level causing major developmental abnormalities in rats is approximately 100 mg/kg. No effect on reproduction was observed in a 3-generation rat study.

2,4-D apparently is not very acutely toxic to humans, with the average oral dose likely to be fatal estimated to be 400 mg/kg. However, considerable uncertainty exists regarding what is a minimal toxic dose; it may be as low as 80 mg/kg. Symptoms of vomiting, fever, and profound muscle weakness are usually reported after ingestion of 2,4-D. 2,4-D is irritating to the eyes. Absorption through the skin reportedly produces severe peripheral neuropathy, with stiffness of extremities, possible motor paralysis, and parathesia.

The oral LD_{50} for 2,4-D in mice and rats is 375 mg/kg, but the oral LD_{50} for dogs is 100 mg/kg. Esters of 2,4-D have comparable toxicity. Cardiac arrhythmia has been cited as a cause of death in several acute studies. Pathological changes have also occurred in the gastrointestinal tract, liver, lungs, and kidneys. The rabbit dermal LD_{50} is 1,400 mg/kg.

Contrary to suggestions that 2,3,7,8-tetrachlorodibenzo-p-dioxin contamination has contributed to the toxicity of 2,4-D, no actual TCDD contamination of 2,4-D has been reported, although hexachlorodibenzo-p-dioxin and 2,7-dichlorodibenzo-p-dioxin have been found. There is no experimental evidence that dioxins are formed by photolysis of 2,4-D.

Toxicity to Wildlife and Domestic Animals

Studies on the effects of exposure to 2,4-D and other phenoxy herbicides on algae indicate that many single-celled plants are not very sensitive to these compounds. Concentrations of 25 mg/liter 2,4-D administered for 10-12 days reduced the growth rate of Scenedesmus, one of the more sensitive species, by 42%. The growth of Nostol muscorum, a blue-green algae, is inhibited at concentrations of 0.1 mg/liter. Various forms of filamentous algae, i.e., Chara, Hydrodictyon, and Pitophora, are controlled at concentrations above 10 mg/liter.

The 96-hour LD_{50} for Daphnia magna is 2 mg/liter. Concentrations of 2 mg/liter had no detectable effect on shell growth in oysters.

2,4-D's toxicity to fish has been thoroughly studied. The 24- and 48-hour LC_{50} values for the bluegill were reported to be 8 mg/liter for 2,4-D. Esters of 2,4-D are slightly more toxic. Concentrations of 50 mg/liter had no observable effect on tadpoles of the frog, Rana temporaria.

Animal poisonings have been reported and attributed to herbicide formulations containing 2,4-D, but in most instances a definite causal relationship has not been established. 2,4-D does not bioaccumulate in the adipose tissue.

Regulations and Standards

OSHA Standard: 10 mg/m^3 TWA

ACGIH Threshold Limit Value: 10 mg/m^3 TWA

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Summary

1,2-Dichloropropane increased the incidence of combined adenomas and carcinomas of the liver when administered to rats and mice, and it was found to be mutagenic using the Ames assay. High concentrations can depress the central nervous system and adversely affect the liver, kidneys, adrenals, and heart.

CAS Number: 78-87-5

Chemical Formula: $CH_2ClCHClCH_3$

IUPAC Name: 1,2-Dichloropropane

Important Synonyms and Trade Names: Propylenechloride, propylenedichloride

Chemical and Physical Properties

Molecular Weight: 112.99

Boiling Point: 96.8°C

Melting Point: -100°C

Specific Gravity: 1.16 at 20°C

Solubility in Water: 2,700 mg/liter at 20°C

Solubility in Organics: Miscible with organic solvents

Log Octanol/Water Partition Coefficient: 2.28

Vapor Pressure: 42 mm Hg at 20°C

Vapor Density: 3.9

Flash Point: 21°C (open cup)

Transport and Fate

Volatilization and subsequent photooxidation are probably important environmental fate processes for 1,2-dichloropropane. In surface water and soil, hydrolysis may also be a significant fate process, especially if the compound is adsorbed onto clay particles. Soil microbes can biodegrade 1,2-dichloropropane, but this is likely to occur more slowly than volatilization. 1,2-Dichloropropane is probably only moderately persistent in the environment.

Health Effects

1,2-Dichloropropane caused an increased incidence of combined adenomas and carcinomas of the liver in male and female mice and caused a slight increase in mammary adenocarcinomas in female rats (NTP 1984). In an earlier study, 80 C3H mice were exposed to 1,850 mg/m^3 of 1,2-dichloropropane for 4 to 7 hours per day 37 times and were then observed for the next 7 months; only 3 mice survived, but all of these developed multiple hepatomas (Heppel et al. 1948). 1,2-Dichloropropane was found to be mutagenic using the Ames assay both with and without metabolic activation. It also increased the frequency of 8 azaguanine-resistant mutants in the _Aspergillus nidulans_ spot test. No information was available on the reproductive or teratogenic effects of this compound.

High concentrations of 1,2-dichloropropane cause central nervous system depression and narcosis in humans. Other human symptoms include headache, vertigo, lacrimation, and irritation of the mucous membranes. Studies indicate that exposure to high concentrations may affect the rate of growth in rats and guinea pigs, and cause fatty degeneration and multilobular or centrilobular necrosis of the liver. Histopathological changes were also observed in the kidneys, adrenals, and heart. 1,2-Dichloropropane is a mild skin irritant. It is moderately irritating to the eye but does not cause permanent injury.

The oral LD_{50} for rats is 1,900 mg/kg; the oral LD_{50} for mice is 860 mg/kg. The dermal LD_{50} for rabbits is 8,750 mg/kg.

Toxicity to Wildlife and Domestic Animals

Only limited data are available on the effects of 1,2-dichloropropane on wildlife and domestic animals. The 48-hour EC_{50} is 52 mg/liter in _Daphnia magna_. The 96-hour EC_{50} for the bluegill is 300 mg/liter; for the fathead minnow, it is 139.3 mg/liter; and for the tidewater sliverside it is 240 mg/liter. In an embryo-larval test using the fathead minnow, chronic effects developed at 8,100 µg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

> The available data are not adequate for establishing criteria.

OSHA Standard (air): 350 mg/m^3 TWA

ACGIH Threshold Limit Values: 350 mg/m^3 TWA
510 mg/m^3 STEL

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1,3-DICHLOROPROPENE

Summary

1,3-Dichloropropene is moderately persistent in soils but less persistent in water. No complete carcinogenicity studies are currently available, but cis-1,3-dichloropropene caused injection-site sarcomas in mice and was found to be mutagenic using the Ames assay. Chronic exposure caused liver and kidney damage.

CAS Number: 542-75-6

Chemical Formula: $CHClCHCH_2Cl$

IUPAC Name: 1,3-Dichloro-1-propene

Important Synonyms and Trade Names: 1,3-Dichloropropylene, Telone, DCP

Chemical and Physical Properties

Molecular Weight: 110.97

Boiling Point: 104°C (cis)
112°C (trans)

Melting Point: No available data

Specific Gravity: 1.217 at 20°C (cis)
1.224 at 20°C (trans)

Solubility in Water: 2,700 mg/liter at 25°C

Solubility in Organics: Soluble in ether, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 1.98

Vapor Pressure: 28 mm Hg at 25°C

Vapor Density: 3.83

Transport and Fate

1,3-Dichloropropene (DCP) is moderately persistent in soils but less persistent in water. Volatilization from soil and water into the atmosphere, where it is subsequently degraded by photooxidation, is probably the predominant transport and fate mechanism for DCP. Although no information was found on the adsorption of 1,3-dichloropropene in aquatic systems, sorption to soil organics is an important terrestrial process and accounts for the persistence of DCP in soils. Hydrolysis of adsorbed material in soil and water to produce 3-chloroallyl alcohol occurs rather slowly but may be an important fate process. Biodegradation of DCP also occurs slowly, but soil bacteria are probably responsible for the degradation of the 3-chloroallyl alcohol to carbon dioxide and water.

Health Effects

A carcinogenicity bioassay of 1,3-dichloropropene is being conducted by the National Toxicology Program. No carcinogenicity studies are currently available, but cis-1,3-dichloropropene caused application-site sarcomas in mice following subcutaneous injection, and the chemical was mutagenic in the Ames assay. No information on the teratogenicity or reproductive toxicity of 1,3-dichloropropene was found in the literature reviewed. Chronic exposure caused liver and kidney toxicity at doses as low as 13.6 mg/m^3. However, after a 3-month recovery period, exposed animals showed no ill effects. The oral LD_{50} value in the rat is 250 mg/kg.

Toxicity to Wildlife and Domestic Animals

Acute LC_{50} values for aquatic organisms exposed to 1,3-dichloropropene were about 6,000 µg/liter in two freshwater species, 1,770 µg/liter in a saltwater fish, and 790 µg/liter in a saltwater invertebrate. Only one chronic toxicity test on 1,3-dichloropropene was reported. This indicated that dichloropropene was toxic at levels of 244 µg/liter to a freshwater fish species.

No information on the toxicity of 1,3-dichloropropene to terrestrial wildlife or domestic animals was found in the sources reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 6,060 µg/liter
Chronic toxicity: 244 µg/liter

Saltwater

Acute toxicity: 790 µg/liter
Chronic toxicity: No available data

Human Health

Criterion: 87 µg/liter

ACGIH Threshold Limit Values: 5 mg/m^3 TWA
50 mg/m^3 STEL

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Summary

Dicofol (Kelthane) is an organochlorine pesticide that is moderately persistent in the environment. In an NCI carcinogenicity bioassay, it produced hepatocellular carcinomas in male mice, but not in rats or female mice. Dicofol caused anomalies in the third-generation offspring in one study on mice but not in another. It also had reproductive effects in rats and mice. Chronic exposure produced liver lesions in rats.

CAS Number: 115-32-2

Chemical Formula: $C_{14}H_9OCl_7$

IUPAC Name: 2,2,2-Trichloro-1,1-bis(4-chlorophenyl)ethanol

Important Synonyms and Trade Names: Kelthane, Acarin, Mitigan

Chemical and Physical Properties

Molecular Weight: 441.5

Boiling Point: Not available in literature reviewed

Melting Point: 79°C

Specific Gravity: 1.130 at 20°C

Solubility in Water: 590 µg/liter at 20°C

Solubility in Organics: Soluble in most aliphatic and aromatic solvents

Log Octanol/Water Partition Coefficient: 5.56

Flash Point: 49°C (closed cup)

Transport and Fate

Little information was found on the transport and fate of dicofol in the environment. It is one of the less persistent organochlorine pesticides and disappears fairly readily from soil. However, trace amounts will persist for up to 1 year. Volatilization is probably the primary transport mechanism, as movement through soil is probably limited by low water solubility and binding to soil material. Based on results for

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DDT, dicofol is probably biodegraded by soil bacteria, and this may be the most important fate process in soil. In aquatic systems, sorption to sediments is probably an important fate, and bioaccumulation may also be important.

Health Effects

Dicofol caused hepatocellular carcinomas in male mice but not in female mice nor in rats of either sex (NCI 1978). In a three-generation study, continuous feeding of 7 ppm (approximately 0.8 mg/kg) dicofol to mice resulted in anomalies in offspring of the third generation. However, in a five-generation study, Brown (1971) noted no fetal anomalies in mice fed up to 500 ppm (approximately 60 mg/kg) dicofol in their diets. Only litter size, weight, and viability decreased significantly at this concentration. Brown (1971) also reported that rats did not produce offspring if fed more than 100 ppm (approximately 5 mg/kg) in their diets. Dicofol was not mutagenic in several microbial test systems.

In a 2-year study in rats, Smith et al. (1959) noted that liver lesions were seen in animals administered more than 1,000 ppm (approximately 45 mg/kg) in their diets and that growth decreased in animals fed diets containing more than 500 ppm (approximately 23 mg/kg) dicofol. The acute oral LD_{50} in rats was 800 mg/kg for males and 680 mg/kg for females.

Toxicity to Wildlife and Domestic Animals

The LC_{50} for rainbow trout exposed to dicofol for 48 hours was 100 mg/liter. The 48-hour LC_{50} values for *Daphnia magna* and stone flies were 390 and 3,000 mg/liter, respectively. Fathead minnows bioconcentrate dicofol to a level 10,000 times that found in water, with a steady-state concentration occurring in 40 to 60 days (Eaton et al. 1983).

The toxicity of dicofol has been studied in several wild bird species. The LC_{50} values for 2-week-old birds fed contaminated feed for 5 days were 1,500 ppm for coturnix, 3,000 ppm for bobwhites, 2,300 for pheasants, and 1,900 for mallard ducks. No information was found on the toxicity of dicofol to other terrestrial wildlife or to domestic animals.

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DIETHYL PHTHALATE

Summary

Diethyl phthalate (DEP) has not been shown to be carcinogenic, but it was found to be mutagenic using bacterial test systems. Intraperitoneal administration to pregnant rats induces adverse reproductive effects. The chronic toxicity of diethyl phthalate is low.

CAS Number: 84-66-2

Chemical Formula: $C_{12}H_{14}O_4$; $C_6H_4(COOC_2H_5)_2$

IUPAC Name: Diethyl ester phthalic acid

Chemical and Physical Properties

Molecular Weight: 222.24

Boiling Point: 298°C

Melting Point: -40.5°C

Specific Gravity: 1.1175 at 20°C

Solubility in Water: 896 mg/liter at 25°C

Solubility in Organics: Soluble in acetone and benzene; miscible with alcohol, ether, ketones, and esters

Log Octanol/Water Partition Coefficient: 3.22 (calculated)

Vapor Pressure: 0.05 mm Hg at 70°C

Flash Point: 162.78°C

Transport and Fate

Much of the information concerning the environmental movement and fate of diethyl phthalate (DEP) is derived from data for phthalate esters in general. DEP probably hydrolyzes in surface waters, but at such a slow rate that this process is not environmentally significant under most conditions. Photolysis and oxidation do not appear to be important environmental fate processes. Volatilization is not an important environmental transport process for DEP in natural waters. However, there is evidence that it can be slowly volatilized from DEP-containing materials at relatively high temperatures. Consequently, some atmospheric dispersion of DEP due to vaporization during manufacture, use, or waste disposal probably occurs.

Adsorption onto suspended solids and particulate matter, and complexation with natural organic substances are probably the most important environmental transport processes for DEP. The octanol/water partition coefficient for DEP suggests that this compound would be adsorbed onto particulates high in organic matter. This contention is supported by the fact that phthalate esters are commonly found in freshwater and saltwater sediment samples. DEP can be dispersed to aquatic and terrestrial systems by complexation with natural organic substances. It readily interacts with the fulvic acid present in humic substances in water and soil, forming a complex which is very soluble in water.

A variety of unicellular and multicellular organisms take up and accumulate phthalate esters, and bioaccumulation of DEP is considered an important fate process. Biodegradation is also considered an important fate process for DEP in aquatic systems and soil. Because phthalate esters, and presumably DEP, are degraded under most conditions and can be metabolized by multicellular organisms, it is unlikely that long-term bioaccumulation or biomagnification occurs.

Analysis using EPA's Exposure Analysis Modeling System suggests that for DEP, chemical and biochemical transformations will compete favorably in ecosystems with long retention times, such as ponds and lakes. If input of DEP remains constant, its concentration is expected to approach a steady state. If input stops, its concentration is expected to decrease relatively quickly. Transport is the dominant process for DEP in rivers, and the oceans are the ultimate sink in these ecosystems.

Health Effects

There are no reports that DEP is carcinogenic in animals or humans. However, DEP is reported to be mutagenic in bacterial test systems (Seed 1982). Reduced fetal weight, resorptions and dose-related musculoskeletal abnormalities were observed among fetuses from rats exposed intraperitoneally to DEP during gestation.

The acute toxicity for laboratory animals by most routes of administration is very low. Oral, inhalation, and intraperitoneal LD_{50} values of 9,000 mg/kg, 7,510 mg/m^3, and 5,058 mg/kg, respectively, are reported for the rat. The no-effect levels determined from chronic feeding studies of six or more weeks duration are 2,500 mg/kg/day for the rat, and 1,250 mg/kg/day for the dog, with no specific lesion attributable to DEP, and no unusual incidence of tumors. In humans, exposure to heated vapor may produce some transient irritation to the nose, throat, and upper respiratory tract.

Toxicity to Wildlife and Domestic Animals

Acute values for the freshwater species *Daphnia magna* and the bluegill are reported to be 52,100 and 98,200 μg/liter DEP, respectively. Among saltwater species, acute values for the mysid shrimp and the sheepshead minnow are 7,590 and 29,600 μg/liter, respectively. No chronic values are available for freshwater or saltwater species.

In the freshwater alga *Selenastrum capricornutum* 96-hour EC_{50} values for chlorophyll *a* and cell number are 90,300 and 85,600 μg/liter, respectively. In saltwater algae, chlorophyll *a* EC_{50} values ranged from 3,000 μg/liter in *Gymnodinium breve* to 65,500 μg/liter in *Skeletonema costatum*. Cell number EC_{50} values in *G. breve* and *S. costatum* are 33,000 and 85,000 μg/liter, respectively. A bioconcentration factor of 117 is reported for DEP and bluegills after a 21 day exposure. The half-life is between 1 and 2 days.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria for diethyl phthalate or for phthalate esters as a group.

Human Health

Criterion: 350 mg/liter

ACGIH Threshold Limit Values: 5 mg/m^3 TWA
10 mg/m^3 STEL

REFERENCES

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DIISOBUTYL KETONE

Summary

Diisobutyl ketone is mildly irritating to the eyes, nose, throat, and skin in humans. Inhalation exposure to high concentrations increased liver and kidney weights in rats and guinea pigs.

CAS Number: 108-83-8

Chemical Formula: $[(Ch_3)_2CHCH_2]_2CO$

IUPAC Name: 2,6-Dimethyl-4-heptanone

Important Synonyms and Trade Names: Isobutyl ketone

Chemical and Physical Properties

Molecular Weight: 142.2

Boiling Point: 166°C

Melting Point: -41.5°C

Specific Gravity: 0.81 at 20°C

Solubility in Water: 500 mg/liter

Solubility in Organics: Miscible with most organic liquids

Log Octanol/Water Partition Coefficient: 2.8 (calculated)

Vapor Pressure: 1.7 mm Hg at 20°C

Vapor Density: 4.9

Flash Point: 60°C

Transport and Fate

No information on the transport and fate of diisobutyl ketone was found in the literature reviewed. Based on its chemical and physical properties, the compound probably is not very volatile. It may be adsorbed by soil organics and sediment to some degree. Ketones in general react in acidic media to form secondary alcohols. This reaction would probably occur in natural waters with low pH. Ketones are not likely to be very persistent in the environment.

Health Effects

Diisobutyl ketone is not very toxic to humans or laboratory animals. It does not appear to be carcinogenic, mutagenic, or teratogenic. A 3-hour exposure by human volunteers to 290 and 580 mg/m^3 slightly irritated the eyes, nose, and throat. In another study of volunteers, some eye irritation and unpleasant odor was reported at concentrations above 145 mg/m^3. Diisobutyl ketone is also mildly irritating to the skin.

Rats and guinea pigs were exposed by inhalation for 7 hours at varying concentrations. At 1,450 mg/m^3 the liver and kidney weights of female rats increased. Concentrations above 2,030 mg/m^3 caused an increase in liver and kidney weights in both sexes, with mortality occurring at the 9,583 mg/m^3 level. An 8-hour inhalation exposure to 11,615 mg/m^3 killed 5 out of 6 rats. The rat oral LD_{50} is 5,750 mg/kg; the rabbit dermal LD_{50} is 17 g/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of diisobutyl ketone to wildlife and domestic animals was available in the sources reviewed.

Regulations and Standards

NIOSH Recommended Standard (air): 140 mg/m^3 TWA

OSHA Standard (air): 300 mg/m^3 TWA

ACGIH Threshold Limit Value: 150 mg/m^3 TWA

REFERENCES

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DIMETHYLAMINOETHYL METHACRYLATE

Summary

Dimethylaminoethyl methacrylate is irritating to the skin, eyes, and mucous membranes and is a strong lachrymator.

CAS Number: 2867-47-2

Chemical Formula: $CH_2C(CH_3)COOCH_2CH_2N(CH_3)_2$

IUPAC Name: 2-Dimethylaminoethyl-2-methylpropenoate

Important Synonyms and Trade Names: 2-Dimethylaminoethyl methacrylate

Chemical and Physical Properties

Molecular Weight: 157

Boiling Point: 187°C

Specific Gravity: 0.933 at 25°C

Solubility in Water: Soluble in water

Solubility in Organics: Soluble in organic solvents

Vapor Density: 5.4

Flash Point: 74°C (open cup)

Transport and Fate

No information on the transport and fate of dimethylaminoethyl methacrylate was available in the sources reviewed.

Health Effects

Only limited data on the toxicity of dimethylaminoethyl methacrylate were found in the literature searched. The compound is an irritant to the skin, eyes, and mucous membranes, and it is a strong lachrymator. The oral and inhalation LD_{50} values for the rat are 1,750 mg/kg and 620 mg/m^3/4 hours, respectively.

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Toxicity to Wildlife and Domestic Animals

No information on the toxicity of dimethylaminoethyl methacrylate to wildlife and domestic animals was found in the sources reviewed.

Regulations and Standards

No regulations or standards have been established for dimethylaminoethyl methacrylate.

REFERENCES

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1984. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1984

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DIMETHYLANILINE

Summary

Dimethylaniline is a central nervous system depressant, and it can cause tremors, convulsions, slowed respiration, and death due to respiratory paralysis. Acute occupational exposure has caused intense abdominal pain, unconsciousness, and visual disturbances. As little as 50 mg/kg was shown to be lethal in humans.

CAS Number: 121-69-7

Chemical Formula: $C_6H_5N(CH_3)_2$

IUPAC Name: N,N-Dimethylaniline

Important Synonyms and Trade Names: N,N-Dimethylbenzeneamine, dimethylphenylamine, n-phenyldimethylamine

Chemical and Physical Properties

Molecular Weight: 121.18

Boiling Point: 193.1°C

Melting Point: 2.5°C

Specific Gravity: 0.9557 at 20°C

Solubility in Water: Slightly soluble

Solubility in Organics: Soluble in alcohol, chloroform, and ether

Log Octanol/Water Partition Coefficient: 2.62

Vapor Pressure: 1 mm Hg at 29.5°C

Vapor Density: 4.17

Flash Point: 61°C

Transport and Fate

Virtually no information on the environmental transport and fate of dimethylaniline is available. Although some volatilization of this compound can occur, it probably is not a significant transport process. Because it is soluble in organic compounds and has a moderate log octanol/water partition coefficient, adsorption to organic particulates in soil or bed sediments may affect dimethylaniline's transport in environmental media. Some bioaccumulation of this compound may also occur. The available data are not adequate to characterize the importance of biodegradation or other fate processes.

Health Effects

Relatively little information on the toxicity of dimethylaniline is available. There are no reports of carcinogenic, mutagenic, or teratogenic activity by this compound in humans or animals. The physiological effects of dimethylaniline have been compared to those of aniline, although the former is thought to be quantitatively less toxic. Toxic effects may be produced as a result of ingestion, inhalation, or absorption through the skin. Dimethylaniline is reported to induce methemoglobin formation in dogs after single oral doses of 50 mg/kg. This compound is a central nervous system depressant and can cause tremors, weakness, tonic and clonic convulsions, slowed respiration, and death due to respiratory paralysis in animals and man. Acute occupational exposure has caused unconsciousness, visual disturbances, and intense abdominal pain. A lethal human oral dose of 50 mg/kg is reported. The oral LD_{50} value for the rat is 1,410 mg/kg. Dimethylaniline is currently being evaluated by the National Toxicology Program's Toxicology Research and Testing Program.

Toxicity to Wildlife and Domestic Animals

Adequate data to characterize the toxicity to wildlife and domestic animals are not available.

Regulations and Standards

OSHA Standard (skin): 25 mg/m^3 TWA

ACGIH Threshold Limit Values (skin): 25 mg/m^3 TWA
50 mg/m^3 STEL

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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DIMETHYLNITROSAMINE

Summary

In the absence of photolytic degradation, dimethylnitrosamine is probably persistent in the environment. Dimethylnitrosamine is carcinogenic and produces lung, liver, and kidney tumors in rats and mice and liver tumors in several other animal species. It also exhibits transplacental carcinogenicity in animals and is mutagenic and embryotoxic. Both acute and chronic exposure have adverse effects on the liver in humans and experimental animals.

CAS Number: 62-75-9

Chemical Formula: $(CH_3)_2NNO$

IUPAC Name: n-Nitrosodimethylamine

Important Synonyms and Trade Names: n-Methyl-n-nitrosomethanamine, n,n-dimethylnitrosamine, DMN, DMNA, NDMA

Chemical and Physical Properties

Molecular Weight: 74.1

Boiling Point: 151°C

Specific Gravity: 1.0 at 20°C

Solubility in Water: Soluble in all proportions

Solubility in Organics: Soluble in organic solvents, lipids

Log Octanol/Water Partition Coefficient: 0.06 to -0.69

Transport and Fate

The most probable environmental fate of dimethylnitrosamine in aqueous solution appears to be slow photolytic degradation. Furthermore, although supporting data are limited, it has been speculated that hydrogen bonding of dimethylnitrosamine with humic acids or coordination with metal cations produces a photolabile intermediate and could lead to moderately rapid degradation in surface waters. Dimethylnitrosamine has been detected

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in the atmosphere of metropolitan areas and near manufacturing facilities emitting this compound, suggesting that some atmospheric transport can occur. However, it is reported that photolytic degradation in air would be rapid, with a half-life of less than 1 hour. Airborne concentrations in excess of a few parts per billion appear to be unlikely except near sources of direct emissions. There is no evidence to suggest that oxidation or hydrolysis are important environmental fates.

Dimethylnitrosamine is completely miscible in water and is reported to be highly solvated. This information, along with limited experimental data, suggest that volatilization from surface waters is probably not an important process. Dimethylnitrosamine has a log octanol/water partition coefficient near 0; significant sorption by organic particulates is therefore unlikely. Experimental evidence confirms this and further suggests that sorption by clay particulates in wet soil is also unlikely. Because dimethylnitrosamine is completely miscible in water and has a low log octanol/water partition coefficient, bioaccumulation is probably an insignificant process. Although biodegradation in surface waters does not appear to be an important environmental fate, slow degradation of dimethylnitrosamine in sewage and soil is reported to occur. Based on this information, it is likely that in the absence of photolytic degradation dimethylnitrosamine would be very persistent in the environment.

Health Effects

Dimethylnitrosamine is considered to be carcinogenic in many experimental animal species by various routes of exposure. Dose-response relationships have been established in several studies. This compound produces liver, lung, and kidney tumors in some species of mice and rats after oral and inhalation exposure. Increased incidences of liver tumors have also been observed in many other animal species after oral administration. Inhalation exposure in rats has produced tumors of the ethroturbinals and nasal cavity. Although insufficient epidemiologic evidence exists to establish a causative role for dimethylnitrosamine in human carcinogenesis, IARC and other public health organizations recommend that this compound be regarded as a human carcinogen.

Dimethylnitrosamine is mutagenic in many microbial test systems with metabolic activation and in several other in vivo and in vitro test systems. This compound is reported to exhibit transplacental carcinogenicity and to be embryotoxic. No teratogenic effects have been reported. Acute and chronic exposure of humans and experimental animals to dimethylnitrosamine resulted primarily in a variety of hepatotoxic effects. In rats, an oral LD$_{50}$ value of 40 mg/kg and an inhalation LD$_{50}$ value of 37 mg/kg are reported.

Toxicity to Wildlife and Domestic Animals

In crayfish exposed to dimethylnitrosamine in water for 6 months, extensive antennal gland degeneration was observed at 200,000 μg/liter and hyperplasia of hepatopancreas tubular cells at 100,000 μg/liter. Rainbow trout fed dimethylnitrosamine for 52 weeks showed a dose-related increase in hepatocellular carcinoma at doses of 200, 400, and 800 mg/kg. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 0.026.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: The available data for nitrosamines in general indicate that toxic effects occur at concentrations as low as 5,850 μg/liter and would occur at lower concentrations among species that are more sensitive than those tested.

Chronic toxicity: No available data

Saltwater

Acute toxicity: The available data for nitrosamines in general indicate that toxic effects occur at concentrations as low as 3,300,000 μg/liter and would occur at lower concentrations among species that are more sensitive than those tested.

Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various levels of dimethylnitrosamine in water are:

Risk	Concentration
10^{-5}	14 ng/liter
10^{-6}	1.4 ng/liter
10^{-7}	0.14 ng/liter

ACGIH Threshold Limit Value: Suspected human carcinogen

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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2,4-DIMETHYLPHENOL

Summary

2,4-Dimethylphenol has been shown to act as a cancer promoter in skin-painting studies, but it has not been tested for carcinogenicity in a complete bioassay. It is an ATP blocking agent. Other dimethylphenols have been shown to cause pathological changes in the heart, liver, and kidneys.

CAS Number: 105-67-9

Chemical Formula: $(CH_3)_2C_6H_3OH$

IUPAC Name: 2,4-Dimethyl-1-hydroxybenzene

Important Synonyms and Trade Names: m-Xylenol, cresylic acid, 2,4-xylenol

Chemical and Physical Properties

Molecular Weight: 122.2

Boiling Point: 210°C

Melting Point: 27°C

Specific Gravity: 0.956 at 20°C

Solubility in Water: 17 g/liter

Solubility in Organics: Freely soluble in alcohol, chloroform, ether, and benzene

Log Octanol/Water Partition Coefficient: 2.50

Vapor Pressure: 0.06 mm Hg at 20°C

pKa: 10.60

Transport and Fate

Photooxidation is probably the primary mechanism for removal of 2,4-dimethylphenol in clear, aerated surface waters, although metal-catalyzed oxidation, sorption, and biodegradation may also have some effect. In murky, unaerated water, biodegradation is probably the primary fate of 2,4-dimethylphenol, with absorption onto organic materials also being somewhat important. 2,4-Dimethylphenol would be expected to adsorb onto organic material in the soil but because of its water solubility it probably moves readily through soil. However, biodegradation would somewhat limit the amount of chemical able to enter the groundwater.

Health Effects

2,4-Dimethylphenol has been shown to be a cancer promoting agent in skin painting studies on rats but has not been tested for its total carcinogenic potential. No studies on the teratogenicity, reproductive toxicity, or mutagenicity of 2,4-dimethylphenol were found in the literature reviewed. At high doses, other dimethylphenols have been shown to cause pathological changes in the liver, kidneys, and heart. 2,4-Dimethylphenol is known to be an ATP blocking agent. Dermal exposure was more toxic to rats than oral dosing. The reported LD_{50} values for the rat were 1,040 mg/kg (dermal) and 3,200 mg/kg (oral).

Toxicity to Wildlife and Domestic Animals

No signs of acute toxicity attributable to 2,4-dimethylphenol were seen in freshwater species exposed to levels less than approximately 2,000 µg/liter. Chronic toxicity studies indicate that the acute-chronic ratio is probably between 5 and 10. The bioconcentration factor in bluegills exposed to 2,4-dimethylphenol for 28 days was 150, but a half-life in the body of less than one day suggests that residues are probably not a significant hazard for freshwater species. No information on the toxicity of 2,4-dimethylphenol to other wildlife or domestic animals was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 2,120 µg/liter
Chronic toxicity: No available data

Acute toxicity: No available data
Chronic toxicity: No available data

Human Health

Health criterion: No available data
Organoleptic criterion: 400 μg/liter

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Summary

n-Dioctyl phthalate (DOP) was fetotoxic and caused developmental abnormalities in one study in rats. It is a severe eye irritant and a mild skin irritant in rabbits.

CAS Number: 117-84-0

Chemical Formula: $C_6H_4(COOC_8H_{17})_2$

IUPAC Name: Di-n-octyl phthalic acid

Important Synonyms and Trade Names: o-Benzenedicarboxylic acid, dioctyl ester, phthalic acid, dioctyl ester, DOP, octyl phthalate

Chemical and Physical Properties

Molecular Weight: 391.0

Boiling Point: 220°C at 5 mm Hg

Melting Point: -25°C

Specific Gravity: 0.978

Solubility in Water: 3 mg/liter at 25°C

Log Octanol/Water Partition Coefficient: 9.2

Vapor Pressure: Less than 0.2 at 150°C

Transport and Fate

Although relatively little specific information concerning n-dioctyl phthalate (DOP) is available, the environmental transport and fate of this compound can be largely inferred from data for phthalate esters as a group. DOP probably hydrolyzes in surface waters, but at such a slow rate that this process would not be significant under most conditions. Photolysis and oxidation do not appear to be important environmental fate processes. Some atmospheric dispersion of DOP that is vaporized during manufacture, use, or disposal can occur. However, volatilization does not appear to be a significant transport process, especially in aquatic systems.

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Adsorption onto suspended solids and particulate matter, and complexation with natural organic substances are probably the most important environmental transport processes for DOP. The high log octanol/water partition coefficient for this compound suggests that it would be readily adsorbed onto particulates high in organic matter. This contention is supported by the fact that phthalate esters are commonly found in freshwater and saltwater sediment samples. DOP can be dispersed through aquatic and terrestrial systems by complexation with natural organic materials. It readily interacts with the fulvic acid present in humic substances in water and soil, forming a complex that is very soluble in water.

A variety of unicellular and multicellular organisms take up and accumulate DOP, and bioaccumulation is considered an important fate process. Biodegradation is also an important fate process in aquatic systems and soil. DOP is biodegraded under most environmental conditions, and it can be metabolized by multicellular organisms. It is unlikely that long-term bioaccumulation or biomagnification occurs.

Analysis based on EPA's Exposure Analysis Modeling System indicates that chemical and biochemical transformation processes for DOP are slow and that transport processes will predominate both in ecosystems that have long retention times (ponds, lakes) and those that have short retention times (rivers). If the input of DOP remains constant, its concentration is expected to increase in aquatic ecosystems. If input stops, the DOP present is expected to persist for an undetermined length of time. The oceans are the ultimate sink for DOP introduced into unimpeded rivers.

Health Effects

There is no evidence to suggest that DOP is carcinogenic or mutagenic. Fetotoxicity and developmental abnormalities were observed in the offspring of rats administered 5 g/kg intraperitoneal injections on days 5 to 15 of gestation. No other evidence for reproductive or teratogenic effects has been reported.

Very little information exists concerning the chronic and acute toxicity of DOP. A chronic LD_{50} value of 1.3 mg/kg was determined for mice receiving intraperitoneal injections of DOP 5 days/week for 10 weeks. DOP has a relatively low acute toxicity in mice with reported oral and intraperitoneal LD_{50} values of 6.5 and 65 g/kg, respectively. This chemical is a severe eye irritant and a mild skin irritant in rabbits.

Toxicity to Wildlife and Domestic Animals

Seven to eight-day LC_{50} values for freshwater species range from 690 to 42,000 µg/liter. A 26-day LC_{50} value of 149,200 µg/liter was reported for rainbow trout.

Freshwater snails and mosquito larvae were found to have bioconcentration factors of 13,600 and 9,400, respectively, in model ecosystems. The bioconcentration factor for a freshwater alga is 28,500.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data are not adequate for establishing criteria.

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1,4-DIOXANE

Summary

1,4-Dioxane caused tumors of the liver and nasal cavity in rats and liver and gall bladder tumors in guinea pigs. IARC has classified dioxane as a potential human carcinogen. Dioxane irritates the eyes and mucous membranes, and inhalation of high concentrations causes liver and kidney damage and edema of the lungs and brain.

CAS Number: 123-91-1

Chemical Formula: $O(CH_2-CH_2)_2O$

IUPAC Name: 1,4-Dioxane

Important Synonyms and Trade Names: p-Dioxane, glycol ethylene ether, 1,4-diethylenedioxyde

Chemical and Physical Properties

Molecular Weight: 88.20

Boiling Point: 101°C

Melting Point: 10°C

Specific Gravity: 1.033 at 20°C

Solubility in Water: Soluble in water

Solubility in Organics: Soluble in organic solvents

Log Octanol/Water Partition Coefficient: -0.42

Vapor Pressure: 30 mm Hg at 20°C

Vapor Density: 3.03

Flash Point: 5°C to 18°C

Transport and Fate

The limited information found on the transport and fate of 1,4-dioxane in the environment suggests that this compound is rather nonreactive. Dioxane would be expected to evaporate slowly; but once in the atmosphere, it should form explosive

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peroxides. It is metabolized by animals to beta-hydroxyethoxyacetic acid and may be biodegraded in a similar fashion by microorganisms. However, no data on biodegradation were available.

Health Effects

1,4-Dioxane has produced malignant tumors of the liver and nasal cavity in rats after chronic exposure and tumors of the liver and gall bladder in guinea pigs after long-term oral administration. In a two-stage carcinogenesis study performed on Swiss-Webster mice, it was also determined that dioxane is a promoter. However, tumors did not develop when rats were exposed to 1,4-dioxane by inhalation. On the basis of the animal studies, it has been concluded that 1,4-dioxane is a potential human carcinogen (IARC 1976). Dioxane has been found to damage DNA, and the results of an in vivo DNA synthesis test were positive. The evidence on the teratogenic potential of dioxane is inconclusive.

Dioxane reportedly irritates the eyes, nose, and throat of humans exposed to concentrations of 1,080 mg/m^3 or more for 15 minutes. Prolonged exposure to concentrations above 1,690 mg/m^3 has caused death, with signs of kidney damage, anemia, and liver necrosis.

In inhalation studies, mice, rabbits, rats, and guinea pigs were exposed to concentrations above 14,400 mg/m^3. Hyperemia and edema of the lungs and brain, in addition to liver and kidney damage, were reported. Experiments with animals indicate that dioxane is not appreciably irritating to intact skin, but it is readily absorbed and causes defatting of the skin layers. The acute LD_{50} values are 5.7 g/kg body weight for mice, 5.2 g/kg body weight for rats, and 3.9 g/kg body weight for guinea pigs. The dermal LD_{50} for rabbits is 7.6 g/kg. Several studies indicate that dioxane may act synergistically with other chemicals.

Toxicity to Wildlife and Domestic Animals

The data on the toxicity of 1,4-dioxane to wildlife and domestic animals are limited. The 96-hour LC_{50} for the bluegill is more than 10,000 mg/liter, and it is 6,700 mg/liter for the tidewater silverside, a saltwater fish. The threshold for inhibition of cell division of the alga *Microcystis aeruginosa* is 575 mg/liter; the threshold for inhibition of cell division in the bacterium *Pseudomonas putida* is 2,700 mg/liter.

Regulations and Standards

NIOSH Recommended Standard: 4 mg/m^3/30 min Ceiling Limit

OSHA Standard (skin): 360 mg/m^3 TWA

ACGIH Threshold Limit Value: 90 mg/m^3 TWA

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DIPHENYLETHANE

Summary

Diphenylethane was shown to be moderately toxic in mice after acute exposure.

CAS Number: 103-29-7

Chemical Formula: $(C_6H_5)CH_2CH_2(C_6H_5)$

IUPAC Name: 1,2-Diphenylethane

Important Synonyms and Trade Names: Bibenzyl, dibenzyl, and 1,2-diphenylethane

Chemical and Physical Properties

Molecular Weight: 182

Boiling Point: 284°C

Melting Point: 52°C

Specific Gravity: 0.978

Solubility in Water: Insoluble in water

Solubility in Organics: Soluble in alcohol, chloroform, ether and carbon disulfide

Log Octanol/Water Partition Coefficient: 4.9 (calculated)

Transport and Fate

No information on the transport and fate of diphenylethane was available in the sources reviewed. Based on its log octanol/water partition coefficient, diphenylethane is probably adsorbed by the organics in soil and sediment. Its ultimate fate in the environment is likely to be either photooxidation or biodegradation by soil microbes.

Health Effects

The available data on the toxicity of diphenylethane was extremely limited. The intraperitoneal and intravenous LD_{50} values for mice, which are 2,500 mg/kg and 78 mg/kg, respectively, were the only values reported.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of diphenylethane to wildlife and domestic animals was found in the sources reviewed.

REFERENCES

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Summary

Endrin is a cyclodiene insecticide that is an isomer of dieldrin. It is probably retained in soils and sediments and is persistent in the environment. It is strongly bioaccumualted by aquatic organisms. Endrin is highly toxic to mammals, aquatic organisms, and terrestrial wildlife after acute exposure. It has not been shown to be carcinogenic or mutagenic, but it is a potent teratogen and reproductive toxin.

CAS Number: 72-20-8

Chemical Formula: $C_{12}H_8Cl_6O$

IUPAC Name: 1,2,3,4,10,10-Hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a octahydro-endo-1,4:5,8-dimethanonaphthalene

Important Synonyms and Trade Names: Endrex, hexadrin, mendrin

Chemical and Physical Properties

Molecular Weight: 380.9

Melting Point: Decomposes at 235°C

Specific Gravity: 1.65 at 25°C

Solubility in Water: 250 μg/liter at 25°C

Solubility in Organics: Soluble in acetone, benzene, carbon tetrachloride, hexane, and xylene

Log Octanol/Water Partition Coefficient: 5.6

Vapor Pressure: 2.7×10^{-7} mm Hg at 25°C

Transport and Fate

Endrin is quite persistent in the environment. Volatilization from soil surfaces and probably from surface water is an important transport process (Nash 1983). Subsequent photolysis to delta-keto endrin and endrin aldehyde are apparently important fate processes. No information on the ability of endrin to adsorb to soils and sediments was found in the literature reviewed, but the physical properties of the chemical suggest that sorption would be an important fate process. Endrin is readily bioconcentrated by aquatic organisms, with concentration factors of 10^3 to 10^4. Biotransformation and biodegradation may also be important fate processes for endrin.

Health Effects

Endrin has not been shown to be carcinogenic or mutagenic. However, it is a potent reproductive toxin and teratogen in experimental animals. Reproductive effects included fetal mortality and growth retardation, while teratogenic effects included cleft palate, open eye, clubbed foot, meningoencephales, and fused ribs. Chronic exposure to low levels of endrin primarily results in nervous system damage but also has adverse effects on the heart, lungs, liver, and kidneys. The acute toxicity of endrin is due to its effects on the central nervous system. The acute oral and dermal LD_{50} values for endrin to the rat were both approximately 15 mg/kg.

Toxicity to Wildlife and Domestic Animals

Endrin is very toxic to aquatic organisms. Freshwater fish were generally more sensitive than invertebrates, with species mean acute values ranging from 0.15 to 2.1 µg/liter. LC_{50} values for saltwater organisms ranged from 0.037 to 14.2 µg/liter. Final acute values for freshwater and saltwater species were 0.18 µg/liter and 0.037 µg/liter, respectively. An acute-chronic ratio of 4.0 was determined from chronic tests on freshwater and saltwater species. Therefore, the freshwater final chronic value was calculated to be 0.045 µg/liter and the saltwater final chronic value was determined to be 0.0093 µg/liter.

Endrin is acutely toxic to terrestrial wildlife and domestic animals and has been used as a rodenticide and an avicide. It can also cause central nervous system effects and reproductive disorders following chronic exposure. Sublethal effects observed in animals exposed to endrin include abnormal behavior, increased postnatal mortality, and increased fetal death.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 0.18 μg/liter
Chronic toxicity: 0.0023 μg/liter

Saltwater

Acute toxicity: 0.037 μg/liter
Chronic toxicity: 0.0023 μg/liter

Human Health

Criterion: 1.0 μg/liter

Primary Drinking Water Standard: 1.0 μg/liter

OSHA Standard: 100 $\mu g/m^3$ TWA

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ETHANOL

Summary

Ethanol is probably responsible for some of the increased risk of cancer associated with the consumption of alcoholic beverages, and it has been found to be mutagenic using several assays. Alcohol consumption has also been associated with a number of teratogenic and reproductive effects and with liver cirrhosis and irritation of the mucous membranes.

CAS Number: 64-17-5

Chemical Formula: C_2H_5OH

IUPAC Name: Ethanol

Important Synonyms and Trade Names: Ethyl alcohol, grain alcohol

Chemical and Physical Properties

Molecular Weight: 46

Boiling Point: 78.4°C

Melting Point: -114.1°C

Specific Gravity: 0.789 at 20°C

Solubility in Water: Miscible in water

Solubility in Organics: Soluble in alcohol, benzene, and ether

Log Octanol/Water Partition Coefficient: -0.31

Vapor Pressure: 44 mm Hg at 20°C

Vapor Density: 1.59

Flash Point: 14°C (closed cup)

Transport and Fate

No information on the transport and fate of ethanol was found in the sources reviewed. However, based on the general reactions of alcohols and the specific chemical and physical properties of the material, likely transport and fate processes can be determined.

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Alcohols are very soluble in water and therefore probably are not very volatile. Some evaporation is likely to occur, however, especially for a compound such as ethanol with a relatively high vapor pressure. Oxidation is probably an important fate process in both surface water and the atmosphere. In soil, ethanol is probably biodegraded by soil microorganisms.

Health Effects

The consumption of alcoholic beverages has been associated with the development in humans of cancer of the esophagus, stomach, colon, and rectum. Excessive consumption of alcohol also appears to act synergistically with smoking to increase the risk of cancer of the mouth, larynx, esophagus, and respiratory tract. Alcohol abuse causes liver cirrhosis, which may in turn lead to hepatomas. Although it appears that at least some of the cancers associated with alcohol consumption may be due to constituents other than ethanol, ethanol is probably responsible for some of the increased risk of cancer. Ethanol was found to be mutagenic in several genotoxicity assays. A number of reproductive and teratogenic effects are associated with alcohol consumption. These include growth deficiencies, delayed motor development, cardiac anomalies, and mental deficiency. Peterson et al. (1981) gave intraperitoneal doses of 6,000 mg/kg daily to pregnant mice on days 6 to 17 of gestation and noted increased resorption and an increase in the incidence of cleft palates.

Excessive ethanol consumption causes liver cell damage and cirrhosis of the liver, as well as the well-known behavioral effects. Ethanol is also an irritant to the mucous membranes. The oral LD_{50} in rats was reported to be 7,060 mg/kg.

Toxicity to Wildlife and Domestic Animals

The 24-hour LD_0 and LD_{100} values for the creek chub were 7,000 and 9,000 mg/liter of ethanol, respectively. The algae Chlorella pyrenoidosa had an LC_{50} of 27,000 mg/liter.

No information on the toxic effects of distilled ethanol to terrestrial wildlife or domestic animals was found in the literature reviewed. However, both terrestrial wildlife and domestic animals have been known to become intoxicated after the consumption of fermented fruit or grain.

Regulations and Standards

OSHA Standard (air): 1,900 mg/m^3 TWA

ACGIH Threshold Limit Value: 1,900 mg/m^3 TWA

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ETHANOLAMINE

Summary

Ethanolamine caused liver and kidney changes when administered orally to rats and liver, lung, and kidney lesions when administered by inhalation to mice, rats, rabbits, and guinea pigs. In humans, it has an irritant and necrotic effect on the skin and mucous membranes, and it is a strong eye irritant.

CAS Number: 141-43-5

Chemical Formula: $NH_2CH_2CH_2OH$

IUPAC Name: Ethanolamine

Important Synonyms and Trade Names: Monoethanolamine, 2-aminoethanol, β-amino ethyl alcohol, ethylolamine, β-hydroxyethylamine, MEA

Chemical and Physical Properties

Molecular Weight: 61.1

Boiling Point: 170°C

Melting Point: 10.3°C

Specific Gravity: 1.018 at 20°C

Solubility in Water: Completely miscible in water

Solubility in Organics: Soluble in alcohol and chloroform

Log Octanol/Water Partition Coefficient: -1.8 (calculated)

Vapor Pressure: 0.4 mm Hg at 20°C

Vapor Density: 2.1

Flash Point: 95°C (closed cup)

Transport and Fate

No specific information on the transport and fate of ethanolamine was found in the literature reviewed. The high water solubility and low vapor pressure suggest that ethanolamine will not volatilize and will tend to move readily with ground-

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or surface water flow. It is a relatively strong base and will therefore disassociate in acidic media.

Health Effects

No information on the carcinogenicity, mutagenicity, reproductive toxicity, or teratogenicity of ethanolamine was found in the sources reviewed. Subchronic (90 day) administration of oral doses of 320 mg/kg/day in feed had no effect on rats, but doses of 140 mg/kg caused increased liver and kidney weights, and doses of 1,280 mg/kg/day caused histopathological changes in these organs and some deaths. Inhalation exposure of rats, mice, rabbits, and guinea pigs to the high concentrations of a vapor or a mist produced hepatic, pulmonary, and renal lesions. Exposure to 15 mg/m^3 of ethanolamine for 90 days caused skin irritation, a slight weight loss, and slight apathy in dogs.

Ethanolamine has an irritant and necrotic effect on the skin and mucous membranes. It is only slightly less irritating to the eye than ammonia and causes redness and swelling when applied to the skin. The dermal LD_{50} in rats of 1,500 mg/kg is lower than the oral LD_{50} in rats of 2,100 mg/kg.

Toxicity to Wildlife and Domestic Animals

The toxicity of ethanolamine to aquatic organisms is dependent on the pH of the system. Goldfish exposed to ethanolamine at a pH of 10.1 had 24- and 96-hour LC_{50} values of 190 and 170 mg/liter, respectively. Goldfish exposed for 24 hours at a pH of 7 had a reported LC_{50} of greater than 5,000 mg/liter. As most natural waters have a pH less than 7, ethanolamine is not likely to be an important aquatic toxin.

No information on the toxicity of ethanolamine to terrestrial wildlife or domestic animals was available in the literature reviewed.

Regulations and Standards

OSHA Standard (air): 8 mg/m^3 TWA

ACGIH Threshold Limit Values: 8 mg/m^3 TWA
15 mg/m^3 STEL

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ETHYL ACETATE

Summary

Inhalation exposure to high levels of ethyl acetate caused pulmonary edema; hemorrhage of the respiratory tract; leukocytosis; and fatty degeneration of various organs, including the liver. Ethyl acetate is a mild irritant of the eyes and mucous membranes.

CAS Number: 141-78-6

Chemical Formula: $CH_3COOC_2H_5$

IUPAC Name: Ethyl acetate

Important Synonyms and Trade Names: Acetic ether, ethyl acetic acid, ethyl ethanoate, vinegar naphtha

Chemical and Physical Properties

Molecular Weight: 88.1

Boiling Point: 77°C

Melting Point: -83°C

Specific Gravity: 0.902 at 20°C

Solubility in Water: 79,000 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, chloroform, and ether

Log Octanol/Water Partition Coefficient: 1.0 (calculated)

Vapor Pressure: 76 mm Hg at 20°C

Flash Point: -4°C

Transport and Fate

No information on the transport and fate of ethyl acetate was found in the literature reviewed. The chemical has a relatively high vapor pressure, but volatilization will be somewhat limited by its high water solubility. Esters usually undergo slow hydrolysis in acidic media, and hydrolysis to acetic acid and ethanol is probably a major fate pathway for ethyl acetate.

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Reduction to form 2 molecules of ethanol could also be important in a reducing environment.

Health Effects

Ethyl acetate was not found to be carcinogenic in a very limited mouse lung tumor bioassay (Stoner et al. 1973). No information on its mutagenicity or reproductive toxicity was found in the sources reviewed.

Animals exposed by inhalation to high concentrations of the vapor (greater than 6,000 mg/m^3) exhibited pulmonary edema, hemorrhage of the respiratory tract, leukocytosis, and fatty degeneration of various organs, including the liver. Humans exposed to 1,400 mg/m^3 noted mild irritation of the nose, eyes, and throat. The acute oral LD_{50} of ethyl acetate in rats is 6,100 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of ethyl acetate to wildlife or domestic animals was available in the sources reviewed.

Regulations and Standards

OSHA Standard (air): 1,400 mg/m^3 TWA

ACGIH Threshold Limit Value: 1,400 mg/m^3 TWA

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ETHYLBENZENE

Summary

There is some evidence suggesting that ethylbenzene causes adverse reproductive effects in animals. Oral and inhalation exposure caused minor liver and kidney changes in rats. Ethylbenzene is a skin and eye irritant.

CAS Number: 100-41-4

Chemical Formula: $C_6H_5C_2H_5$

IUPAC Name: Ethylbenzene

Important Synonyms and Trade Names: Phenylethane, EB, ethylbenzol

Chemical and Physical Properties

Molecular Weight: 106.2

Boiling Point: 136.2°C

Melting Point: -95°C

Specific Gravity: 0.867 at 20°C (liquid)

Solubility in Water: 161 mg/liter at 25°C

Solubility in Organics: Freely soluble in organic solvents

Log Octanol/Water Partition Coefficient: 3.15

Vapor Pressure: 7 mm Hg at 20°C

Vapor Density: 3.66

Henry's Law Constant: 6.44 atm. m^3/mole

Flash Point: 17.2°C

Transport and Fate

Only limited data are available on the transport and fate of ethylbenzene. Volatilization is probably the major route of elimination from surface water. Subsequent atmospheric reactions, especially photooxidation, are responsible for its

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fate. However, its high log octanol/water partition coefficient suggests that a significant amount of ethylbenzene may be adsorbed by organic material in the sediment. Some soil bacteria are capable of using ethylbenzene as a source of carbon. However, the relative importance of this potential route of ethylbenzene elimination has not been determined.

Health Effects

Ethylbenzene has been selected by the National Toxicology Program to be tested for possible carcinogenicity, although negative results were obtained in mutagenicity assays in *Salmonella typhimurium* and *Saccharomyces cerevisiae*. There is recent animal evidence that ethylbenzene causes adverse reproductive effects. Ethylbenzene is a skin irritant, and its vapor is irritating to the eyes at a concentration of 200 ppm (870 mg/m^3) and above. When experimental animals were exposed to ethylbenzene by inhalation, 7 hours/day for 6 months, adverse effects were produced at concentrations of 600 ppm (2,610 mg/m^3) and above, but not at 400 ppm (1,740 mg/m^3). At 600 ppm rats and guinea pigs showed slight changes in liver and kidney weights, monkeys had slight changes in liver weight, and monkeys and rabbits experienced histopathologic changes in the testes. Similar effects on the liver and kidney were observed in rats fed ethylbenzene at 408 and 680 mg/kg/day for 6 months.

Toxicity to Wildlife and Domestic Animals

Ethylbenzene was acutely toxic to freshwater species at levels greater than 32 mg/liter. No chronic toxicity was reported, but the highest test dose (440 µg/liter) was only one-hundredth of the 96-hour LC_{50} for the particular species being tested. No studies on the bioaccumulation of ethylbenzene were reported in the information reviewed, but a bioconcentration factor of 95 was calculated using the log octanol/water partition coefficient. No information on the toxicity of ethylbenzene to domestic animals and terrestrial wildlife was found in the sources reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing final criteria. However, EPA did report the lowest values known to have toxic effects in aquatic organisms.

Freshwater

Acute toxicity: 32,000 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 430 μg/liter
Chronic toxicity: No available data

Human Health

Criterion: 1.4 mg/liter

OSHA Standard (skin): 435 mg/m^3 TWA

ACGIH Threshold Limit Values: 435 mg/m^3 TWA
545 mg/m^3 STEL

REFERENCES

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ETHYLENE AND DIETHYLENE GLYCOL

Summary

Ethylene and diethylene glycol produce similar toxic effects. Both caused bladder stones, severe kidney damage, and moderate liver damage in rats when administered chronically in the diet. Inhalation exposure causes nausea, throat irritation, and dizziness. Musculoskeletal abnormalities and cranofacial defects were observed in the offspring of pregnant rats given high doses of ethylene glycol orally.

CAS Number: Ethylene glycol: 107-21-1
Diethylene glycol: 111-46-6

Chemical Formula: Ethylene glycol: $C_2H_4(OH)_2$
Diethylene glycol: $O(C_2H_4OH)_2$

IUPAC Name: Ethylene glycol: 1,2-Ethanediol
Diethylene glycol: 2,2-Oxydiethanol

Important Synonyms and Trade Names:

Ethylene glycol: Ethylene alcohol, 1,2-dihydroxy ethane, glycol
Diethylene glycol: bis(2-Hydroxyethyl)ether, diglycol, ethylene diglycol

Chemical and Physical Properties

Molecular Weight: Ethylene glycol: 62
Diethylene glycol: 106

Boiling Point: Ethylene glycol: 197°C
Diethylene glycol: 245°C

Melting Point: Ethylene glycol: -13.5°C
Diethylene glycol: -10°C

Specific Gravity: 1.12 at 20°C

Solubility in Water: Soluble in water

Solubility in Organics: Soluble in alcohol, ether, and acetone

Log Octanol/Water Partition Coefficient:

Ethylene glycol: -2 (calculated)
Diethylene glycol: -1.4 (calculated)

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Vapor Pressure: Ethylene glycol: 0.05 mm Hg at 20°C
Diethylene glycol: <0.01 mm Hg at 20°C

Vapor Density: Ethylene glycol: 2.14
Diethylene glycol: 3.66

Flash Point: Ethylene glycol: 116°C
Diethylene glycol: 124°C

Transport and Fate

The limited information on the transport and fate of ethylene and diethylene glycol suggests that they are unlikely to volatilize and that biodegradation in soil or surface water is probably an important fate process for both compounds. Their high solubilities and low log octanol/water partition coefficients suggest that they move freely in water. Oxidation may be an important fate process in surface water.

Health Effects

The lethal oral dose of ethylene glycol for humans is approximately 1.4 ml/kg, or 100 ml for an adult man weighing 70 kg. Children are apparently less susceptible than adults to ethylene glycol poisoning. Ingestion of the compound can lead to prostration or unconsciousness, accompanied by metabolic acidosis and renal damage.

Inhalation exposure to more than 140 mg/m^3 of ethylene glycol causes irritation of the throat, mild headache, and possibly pain in the lower back. Eye irritation from splashing liquid or exposure to vapor is possible; in one study, workers developed nystagmus when exposed to high levels of ethylene glycol vapor. It has not been established that ethylene glycol is a skin irritant, although transient, mild irritation is possible.

In one chronic study, rats that were fed diets containing 1% or 2% ethylene glycol (approximately 500 to 1,000 mg/kg/day) for 2 years had shorter life spans, developed calcium oxalate bladder stones, and suffered from centrilobular liver degeneration and severe injury to the renal tubules. Ethylene glycol is metabolized in the body to oxalic acid, which is deposited in the kidney as calcium oxalate. The symptoms of ethylene glycol poisoning--metabolic acidosis and renal damage--have been prevented in monkeys by the administration of alcohol dehydrogenase inhibitors.

Ethylene glycol is currently being tested for carcinogenicity by the National Toxicology Program. It has been reported that it inhibits DNA synthesis and causes mutations in cultured mouse lymphocytes. The reproductive and teratogenic effects of ethylene glycol have not been determined conclusively, but craniofacial defects and musculoskeletal abnormalities were reported in rat fetuses when very high doses of ethylene glycol were administered orally to pregnant females.

Ethylene glycol is less toxic to many animal species than it is to humans. For example, the oral LD_{50} for rats is 4.7 g/kg; for mice, 7.5 g/kg; and for guinea pigs, 6.6 g/kg. The dermal LD_{50} for rabbits is 19.5 g/kg. Eye irritation has been reported in rabbits and rats after exposure to airborne concentrations of 12 mg/m^3 for 3 days.

Diethylene glycol causes symptoms similar to those produced by ethylene glycol: nausea, dizziness, and severe kidney damage, followed by oligurea or anuria. The lethal oral dose for humans is approximately 1 ml/kg.

In a 2-year study, diethylene glycol fed to rats at dietary concentrations of 4% (approximately 2,000 mg/kg/day) caused lower growth rates, bladder stones, severe kidney damage, and moderate liver damage. Although there are reports that diethylene glycol causes bladder tumors in rats, these tumors are thought to be the result of mechanical irritation caused by calcium oxalate stones in the bladder. Diethylene glycol has not been reported to be mutagenic. Its reproductive and teratological effects are not known.

The oral LD_{50} for the mouse is 23.7 g/kg, and for the guinea pig it is 7.8 g/kg. Humans are more than 13 times more sensitive to diethylene glycol poisoning than rats. The dermal LD_{50} for rabbits is 11.9 g/kg.

Toxicity to Wildlife and Domestic Animals

Only limited information on the toxicity of ethylene glycol and diethylene glycol to wildlife is available. Concentrations of 250 mg/liter of ethylene glycol are toxic to *Pseudomonas putida*; *Chlorella pyrenoidosa* is killed by 180 mg/liter. The 24-hour LD_{50} for goldfish is more than 5,000 mg/liter. For diethylene glycol, inhibition of cell multiplication in *Pseudomonas putida* starts at 8,000 mg/liter. Toxicity to the alga *Microcystis aeruginosa* starts at 1,700 mg/liter. The 24-hour LD_{50} for goldfish is more than 5,000 mg/liter. Bioaccumulation does not occur.

Regulations and Standards

ACGIH Threshold Limit Value:

Ethylene glycol: 125 mg/m^3 Ceiling Level

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ETHYL ETHER

Summary

Ethyl ether produced adverse reproductive effects in the offspring of pregnant rats and mice after the administration of a single anesthetic dose. It is a mild skin and eye irritant.

CAS Number: 60-29-7

Chemical Formula: $C_2H_5OC_2H_5$

IUPAC Name: Ethoxyethane

Important Synonyms and Trade Names: Diethylether, ethoxyethane, ethyloxide, diethyloxide, sulfuric ether

Chemical and Physical Properties

Molecular Weight: 74.12

Boiling Point: 34.5°C

Melting Point: -116.2°C

Specific Gravity: 0.7138 at 20°C

Solubility in Water: 60,000 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, acetone, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 1.4 (calculated)

Vapor Pressure: 442 mm Hg at 20°C

Vapor Density: 2.56

Flash Point: -45°C

Transport and Fate

No information on the transport and fate of ethyl ether was found in the literature reviewed.

From information on the chemical and physical properties of ether, it appears that volatilization would be an important transport pathway from soil. Ethyl ether reacts slowly with air to form explosive peroxides. High solubility and high vapor density would limit volatilization somewhat and suggest that transport in groundwater may also occur. Ethyl ether has a low log octanol/water partition coefficient and therefore probably is not sorbed to any significant extent.

Health Effects

No information on the carcinogenicity of ether was reported in the literature reviewed. Ethyl ether inhibited DNA repair in an assay using Escherichia coli. Pregnant female rats and mice were anesthetized with ethyl ether for 1 hour either early or late in gestation (Schwetz and Becker 1970). In mice, exposure to ethyl ether during early embryogenesis caused a significant increase in resorptions and decrease in the length of fetal long bones. Early or late exposure caused an increase in the incidence of generalized edema, missing sternum, unossified phalanges, and missing cervical vertebrae. In rats, anesthesia during early or late embryogenesis decreased fetal body weight and the length of the long bones.

Ether at high concentrations (greater than 100 g/m^3) causes narcosis and general anesthesia. It will cause minor skin and eye irritation at 90 mg/m^3. The oral LD_{50} of ethyl ether in the rat is 1,215 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of ethyl ether to wildlife or domestic animals was found in the literature reviewed.

Regulation and Standards

OSHA Standard (air): 1,200 mg/m^3 TWA

ACGIH Threshold Limit Values: 1,200 mg/m^3 TWA
1,500 mg/m^3 STEL

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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ETHYL HEXANEDIOL

Summary

Ethyl hexanediol is quite irritating to the eyes.

CAS Number: 94-96-2

Chemical Formula: $C_8H_{16}(OH)_2$

IUPAC Name: 2-Ethyl-1,3-hexanediol

Important Synonyms and Trade Names: Carbide 6-12, Cmpd 6-12 insect repellent, ethohexadiol, ethyl hexy lene glycol, 2-ethyl-3-propyl-1,3-propanediol, Rutgers 612

Chemical and Physical Properties

Molecular Weight: 146.26

Boiling Point: 243.1°C

Specific Gravity: 0.9422 at 20°C

Solubility in Water: 6000 mg/liter

Solubility in Organics: Soluble in alcohol, ehter, propylene glycol, and castor oil

Log Octanol/Water Partition Coefficient: 1 (calculated)

Vapor Pressure: <0.01 mm Hg at 20°C

Vapor Density: 5.03

Flash Point: 127°C (open cup)

Transport and Fate

No information on the transport and fate of ethyl hexanediol was available in the sources reviewed. Ethyl hexanediol is fairly soluble in water, has a low vapor pressure, and therefore probably is not volatile. Reactions typical of alcohols, such as oxidation or esterification, are likely to be important in determining the fate of ethyl hexanediol.

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Health Effects

Only limited information was available on ethyl hexanediol in the sources reviewed. The compound is quite irritating to the eye but does not irritate the skin. The oral LD_{50} in the rat is 1,400 mg/kg, and the dermal LD_{50} for the rabbit is 2,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of ethyl hexanediol to wildlife was found in the literature reviewed. An oral LD_{50} of 1,400 mg/kg was determined for the chicken.

REFERENCES

LYMAN, W.J., REEHL, W.F., and ROSENBLATT, D.H. 1982. Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds. McGraw-Hill Book Co., New York

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WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

BIS(2-ETHYLHEXYL)PHTHALATE

Summary

bis(2-Ethylhexyl)phthalate (DEHP) is probably persistent in the environment. It is carcinogenic in rats and mice, causing hepatocellular carcinomas. Teratogenic and reproductive effects have been observed in experimental animals. Chronic exposure to DEHP retarded growth and increased liver and kidney weights in animals.

CAS Number: 117-81-7

Chemical Formula: $C_6H_4(COOCH_2CH(C_2H_5)C_4H_9)_2$

IUPAC Name: bis(2-Ethylhexyl)ester phthalic acid

Important Synonyms and Trade Names: DEHP, Di(2-ethylhexyl)phthalate, bis(2-ethylhexyl)ester phthalic acid

Chemical and Physical Properties

Molecular Weight: 391.0

Boiling Point: 386.9°C at 5 mm Hg

Melting Point: -50°C

Specific Gravity: 0.985

Solubility in Water: 0.4 mg/liter at 25°C

Solubility in Organics: Miscible with mineral oil and hexane

Log Octanol/Water Partition Coefficient: 5.3

Vapor Pressure: 2×10^{-7} mm Hg at 20°C

Flash Point: 218.33°C

Transport and Fate

bis(2-Ethylhexyl)phthalate (DEHP) is the most thoroughly studied of the phthalate esters. It probably hydrolyzes in surface waters, but at such a slow rate that this process is not environmentally significant under most conditions. Photolysis and oxidation do not appear to be important environmental fate processes. Although some researchers suggest that volatilization of DEHP from aqueous solution may be significant under some conditions, it probably is not an important environmental transport process in natural waters. In contrast, there is evidence that this compound can be slowly volatilized from DEHP-containing materials at relatively high temperatures. Consequently, some atmospheric dispersion of DEHP due to vaporization during manufacture, use, or waste disposal probably occurs.

Adsorption onto suspended solids and particulate matter and complexation with natural organic substances are probably the most important environmental transport processes for DEHP. The log octanol/water partition coefficient for DEHP suggests that this compound would be adsorbed onto particulates high in organic matter. This contention is supported by the fact that phthalate esters are commonly found in freshwater and saltwater sediment samples. DEHP can be dispersed from sources of manufacture and use to aquatic and terrestrial systems by complexation with natural organic substances. It readily interacts with the fulvic acid present in humic substances in water and soil, forming a complex that is very soluble in water.

A variety of unicellular and multicellular organisms take up and accumulate DEHP, and bioaccumulation is considered an important fate process. Biodegradation is also considered an important fate process in aquatic systems and soil. DEHP is degraded under most conditions and can be metabolized by multicellular organisms. Therefore, it is unlikely that long-term biomagnification occurs.

Analysis using EPA's Exposure Analysis Modeling System suggests that chemical and biochemical transformation processes for DEHP are slow and that transport processes will predominate both in ecosystems that have long retention times (ponds, lakes) and in those that have short retention times (rivers). If the input of DEHP remains constant, its concentration is expected to increase in aquatic ecosystems. If the input stops, the DEHP present is expected to persist for an undetermined length of time. The oceans are the ultimate sink for DEHP introduced into unimpeded rivers.

Health Effects

DEHP is reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules after oral administration (NTP 1982). Its status as a human carcinogen is considered indeterminate by the International Agency for Research on Cancer (IARC). The results of dominant lethal experiments with mice suggest that

DEHP is mutagenic when injected intraperitoneally. However, most experiments conducted with microorganisms and mammalian cells have failed to demonstrate genotoxic activity. Teratogenic and fetotoxic effects have been observed in experimental animals after oral and intraperitoneal administration. Other reproductive effects, including testicular changes in rats and mice, have also been reported.

DEHP appears to have a relatively low toxicity in experimental animals. The oral, intraperitoneal, and intravenous LD_{50} values reported for DEHP in rats are 31 g/kg, 30.7 g/kg, and 0.25 g/kg, respectively. DEHP is poorly absorbed through the skin, and no irritant response or sensitizing potential from dermal application has been noted in experimental animals or humans.

Chronic exposure to relatively high concentrations of DEHP in the diet has caused retardation of growth and increased liver and kidney weights in experimental animals.

Toxicity to Wildlife and Domestic Animals

Acute median effect values ranged from 1,000 to 11,100 µg/liter DEHP for the freshwater cladoceran *Daphnia magna*. The LC_{50} values for the midge, scud, and bluegill all exceeded the highest concentrations tested, which were 18,000, 32,000, and 770,000 µg/liter, respectively. As these values are greater than the water solubility of the chemical, it is unlikely that DEHP will be acutely toxic to organisms in natural waters. In a chronic toxicity test with *Daphnia magna*, significant reproductive impairment was found at the lowest concentration tested, 3 µg/liter. A chronic toxicity value of 8.4 µg/liter was reported for the rainbow trout. No acute or chronic values were reported for saltwater invertebrates or vertebrates. Reported bioconcentration factors for DEHP in fish and invertebrates range from 14 to 2,680.

Although insufficient data were presented to calculate the acute-chronic ratio for DEHP, it is apparently on the order of 100 to 1,000. Therefore, acute exposure to the chemical is unlikely to affect aquatic organisms adversely, but chronic exposure may have detrimental effects on the environment.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria for bis(2-ethylhexyl)phthalate or for phthalate esters as a group.

Human Health

Criterion: 15 mg/liter

ACGIH Threshold Limit Values: 5 mg/m^3 TWA
10 mg/m^3 STEL

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U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-067

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2332 pages

FLUORANTHENE

Summary

Fluoranthene is a polycyclic aromatic hydrocarbon (PAH). It is probably persistent in the environment. Fluoranthene dos not appear to be a complete carcinogen, but it has been shown to be a potent cocarcinogen in animal test systems.

CAS Number: 206-44-0

Chemical Formula: $C_{16}H_{10}$

Chemical and Physical Properties

Molecular Weight: 202.26

Boiling Point: Approximately 375°C

Melting Point: 111°C

Specific Gravity: 1.252 at 0°C

Solubility in Water: 0.26 mg/liter

Solubility in Organics: Soluble in ethanol, ether, benzene, chloroform, acetic acid, and carbon disulfide

Log Octanol/Water Partition Coefficient: 5.33 (calculated)

Vapor Pressure: 10^{-6} to 10^{-4} mm Hg at 20°C (estimated)

Transport and Fate

Much of the information concerning transport and fate is inferred from data for polycyclic aromatic hydrocarbons (PAHs) in general because of a lack of specific information on fluoranthene. Rapid, direct photolysis of fluoranthene to quinones may occur in aqueous solution. The oxidation of fluoranthene is probably too slow to be a significant environmental process, and the available data suggest that volatilization generally is not an important transport process for fluoranthene. The calculated log octanol/water partition coefficient of 5.33 indicates that the compound should be strongly adsorbed onto particulate matter, especially particulates high in organic content. It is likely that fluoranthene can be transported as adsorbed matter on suspended particulates in air or water. Data for PAHs in general indicate that fluoranthene will accumulate in the sediment and biota of the aquatic environment and that adsorption is probably the dominant aquatic transport process.

Data for a variety of PAHs suggest that bioaccumulation is a short-term process, and long-term partitioning into biota is not a significant fate process. Fluoranthene can be metabolized by multicellular organisms and degraded by microbes. Degradation by mammals is likely to be incomplete; the parent compound and the metabolites are excreted by the urinary system. Biodegradation by microorganisms is probably the ultimate fate process. Biodegradation generally appears to be slower in aquatic systems than in soil. However, it may be important in those aquatic systems that are chronically affected by PAH contamination. Fluoranthene is stable enough in air to be transported over relatively large distances.

Health Effects

There is no information concerning the carcinogenicity of fluoranthene in humans, and fluoranthene shows no activity as a complete carcinogen in experimental animals. However, fluoranthene appears to possess potent cocarcinogenic activity in test animals. Fluoranthene has displayed no mutagenic activity in in vitro bacterial test systems. No other information is available concerning its potential mutagenic or teratogenic effects, nor with regard to its acute or chronic toxicity to humans. Results from animal studies indicate that fluoranthene has relatively low acute toxicity. Where deaths of experimental animals have occurred, no information concerning target organs or specific causes of death has been reported. Descriptions of chronic toxicity are limited to reports of mortality produced in mice by repeated dermal application or subcutaneous injection.

Toxicity to Wildlife and Domestic Animals

Among freshwater species, the bluegill, with a 96-hour LC_{50} value of 3,980 µg/liter, is more sensitive to fluoranthene than the cladoceran *Daphnia magna*, with a 48-hour EC_{50} value of 325,000 µg/liter. No chronic data are available for freshwater organisms. Among saltwater species, the 96-hour LC_{50} values for the mysid shrimp and a polychaete are 40 and 500 µg/liter, respectively. The 96-hour LC_{50} value for the sheepshead minnow is greater than 560,000 µg/liter. The chronic value and acute-chronic ratio for the mysid shrimp are 16 µg/liter and 2.5, respectively. The freshwater and saltwater algal species tested exhibit similar sensitivities to fluoranthene, with EC_{50} values of about 50,000 µg/liter. There is evidence of fluoranthene accumulation in edible aquatic organisms, although no measured, steady-state bioconcentration factors are available for freshwater or saltwater organisms.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest concentrations of fluoranthene known to cause toxic effects in aquatic organisms.

Freshwater

Acute toxicity: 3,980 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 40 μg/liter
Chronic toxicity: 16 μg/liter

Human Health

Criterion: 42 μg/liter

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FORMALDEHYDE

Summary

Formaldehyde has been shown to produce nasal tumors in rats, and there is suggestive evidence that it produces the same type of tumor in humans. Inhalation exposure to formaldehyde causes respiratory irritation and can also produce localized effects in the nose, throat, and lungs. In addition, formaldehyde can irritate the skin and cause allergic dermatitis in susceptible individuals.

CAS Number: 50-00-0

Chemical Formula: CH_2O

IUPAC Name: Methanal

Important Synonyms and Trade Names: Methanal, Formalin (formaldehyde solution)

Chemical and Physical Properties

Molecular Weight: 30.03

Boiling Point: 19.5°C

Melting Point: -92°C

Specific Gravity: 0.867 at 20°C

Solubility in Water: Undergoes solvation in water or methanol

Solubility in Organics: Soluble in chloroform, ether, and toluene

Vapor Pressure: 760 mm Hg at 19.5°C

Vapor Density: 1.075

Flash Point: 300°C

Transport and Fate

Formaldehyde is a gas at ambient temperatures so the air will be a major route of transport. In water, formaldehyde is rapidly hydrated and converted to methylene glycol and polyoxymethylene glycols, however, it can volatilize as formaldehyde.

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Formaldehyde readily adsorbs to clay soils but sorption to soil with smaller amounts of organic material is probably negligible.

Photolysis of formaldehyde occurs in the lower troposphere by two primary processes. One process predominates at wavelengths of 290 to 313 nm and the other predominates at wavelengths of 313 to 360 nm. Reported products of formaldehyde photolysis are formyl radicals and water. The estimated half-life of formaldehyde in sunlight is about 75 minutes. In water, formaldehyde is hydrated to methylene glycol and polyoxymethylene glycols which do not undergo photolysis. Further degradation occurs in water, primarily due to biodegradation.

Health Effects

Formaldehyde has been shown to produce nasal tumors in rats, and there is suggestive evidence that it produces the same type of tumor in humans (Siegel et al. 1983). It has been shown that formaldehyde is a "weak" mutagen producing gene mutations and chromosomal abberrations in a variety of laboratory test systems. Formaldehyde has also caused cell transformation in cell culture systems. Formaldehyde has not been shown to be teratogenic or to cause reproductive toxicity in animal studies. However, the studies have not been adequate to fully assess these toxicities.

Formaldehyde is a respiratory irritant and has been found to produce localized effects in the nose, throat, and tracheobronchial tree of exposed individuals. Irritation of the skin has also been reported. In addition, an allergic dermatitis has been produced in some people exposed to formaldehyde. The inhalation LD_{50} in rats was reported to be 100 mg/m^3.

Toxicity to Wildlife and Domestic Animals

Data concerning toxicity to wildlife and domestic animals are not available.

Regulations and Standards

NIOSH Recommended Standard: 0.6 mg/m^3

OSHA Standard (Air): 3.6 mg/m^3 TWA

ACGIH Threshold Limit Value: 1.5 (Suspect Carcinogen)

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HEPTACHLOR

Summary

Heptachlor is an organochlorine pesticide. Together with its active metabolite, heptachlor epoxide, it is very persistent in the environment. When administered orally to mice, both substances cause liver tumors. They also have mutagenic effects. These chemicals have a number of reproductive and teratogenic effects, including decreased litter size, shortened life span of suckling young, and the development of cataracts in offspring. The acute toxicity of both heptachlor and heptachlor epoxide is very high. Chronic exposure induces liver changes and may cause kidney damage. Heptachlor is also highly toxic to fish and wildlife.

Background Information

Technical heptachlor is a complex mixture containing approximately 72% heptachlor and 28% related compounds. It is a soft wax with a melting point of 46-74°C.

CAS Number: 76-44-8

Chemical Formula: $C_{10}H_5Cl_7$

IUPAC Name: 1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene

Chemical and Physical Properties

Molecular Weight: 373.3

Melting Point: 95-96°C

Specific Gravity: 1.57-1.59 at 9°C

Solubility in Water: 0.056 to 0.180 mg/liter at 25-29°C depending on particle size

Solubility in Organics: Soluble in ethanol, ether, benzene, acetone, carbon tetrachloride, xylene, kerosene, cyclohexanone, and ligroin

Vapor Pressure: 0.0003 mm Hg at 25°C

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Transport and Fate

Heptachlor and its active metabolite, heptachlor epoxide, are very persistent in the environment, resisting chemical and biological breakdown into harmless substances. Sorption of heptachlor to sediments appears to be an important process for removal of the pesticide from water, as residue concentrations in sediment are often much higher than in water. Some volatilization may also occur.

Heptachlor and heptachlor epoxide bind tightly to soil particles and will persist for years in soil after surface application. However, heptachlor applied as an emulsifiable concentrate is more readily volatized than when applied as a granular formulation. Certain crops accumulate residues of these compounds by absorption from the soil.

Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur. Heptachlor and heptachlor epoxide are widespread in ambient air, but generally occur at low concentrations. However, levels vary both geographically and seasonally.

Health Effects

Heptachlor and heptachlor epoxide are liver carcinogens when administered orally to mice. Results from mutagenicity bioassays suggest that these compounds also may have genotoxic activity. Reproductive and teratogenic effects in rats include decreased litter size, shortened life span of suckling rats, and development of cataracts in offspring.

Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects due to heptachlor exposure. Although heptachlor and heptachlor epoxide are absorbed most readily through the gastrointestinal tract, inhalation and skin contact are also potential routes of exposure. Acute exposure by various routes can cause developmment of hepatic vein thrombi and can affect the central nervous system and cause death. Chronic exposure induces liver changes, affects hepatic microsomal enzyme activity, and causes increased mortality in offspring. The oral LD_{50} in the rat is 40 mg/kg for heptachlor and 47 mg/kg for heptachlor epoxide.

Although there are reports of acute and chronic toxicity in humans, with symptoms including tremors, convulsions, kidney damage, respiratory collapse, and death, details of such episodes are not well documented. Heptachlor epoxide has been found in a high percentage of human adipose tissue samples, and also in human milk samples and biomagnification of heptachlor/heptachlor epoxide occurs. This compound also has been found in the tissues of stillborn infants, suggesting an ability to cross the placenta and bioaccumulate in the fetus.

Toxicity to Wildlife and Domestic Animals

Heptachlor is toxic at low concentrations in some aquatic invertebrate and fish species. Heptachlor epoxide appears to be a minor product of heptachlor transformations in aquatic systems but the capability of different organisms to effect epoxidation varies. Mean acute values for freshwater species range from 0.9 to 78 μg/liter for invertebrates and from 13.1 to 320 μg/liter for fish. A life cycle test conducted with the fathead minnow provides a chronic value of 1.26 μg/liter and an acute-chronic ratio of 80 for this species. Saltwater mean acute values range from 0.04 to 194 μg/liter for a variety of fish and invertebrate species. A chronic value of 1.58 μg/liter and an acute-chronic ratio of 3.9 are reported for the sheepshead minnow.

Heptachlor shows a strong tendency to bioaccumule. It can concentrate at levels thousands of times greater than those in the surrounding water in a variety of aquatic organisms. Because of this tendency for bioaccumulation, chronic exposure to levels greater than 0.004 μg/liter is considered potentially harmful to aquatic life. However, this value may be too high because the average concentration in a high lipid species will be at FDA action levels for human consumption.

Heptachlor and heptachlor epoxide residues have been found in a wide variety of wildlife and domestic animal species, but usually at relatively low levels. The use of heptachlor as a seed dressing for cereal grains has been linked to mortality among granivorous birds and to increased residues in the tissues of granivorous birds and mammals. Residues have also been found in raptors but a causal relationship with observed toxic effects has not been established. Increased mortality among birds, mammals, fish, and aquatic species has been reported in areas treated with heptachlor. Heptachlor or heptachlor epoxide residues have regularly been found in food and feed crops, meat, fish, poultry, dairy products, and eggs. Oral LC_{50} values for heptachlor ranging from 92 to 480 ppm in their diet (around 20 mg/kg body weight) are reported for wild bird species.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 0.52 µg/liter
Chronic toxicity: 0.0038 µg/liter

Saltwater

Acute toxicity: 0.053 µg/liter
Chronic toxicity: 0.0036 µg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of heptachlor in water are:

Risk	Concentration
10^{-5}	2.78 ng/liter
10^{-6}	0.28 ng/liter
10^{-7}	0.028 ng/liter

CAG Unit Risk (USEPA): 3.37 $(mg/kg/day)^{-1}$

OSHA Standard (skin): 0.5 mg/m^3 TWA

ACGIH Threshold Limit Values (skin): 0.5 mg/m^3 TWA
2 mg/m^3 STEL

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HEXACHLOROBENZENE

Summary

Hexachlorobenzene is very persistent in the environment and can be bioaccumulated. It is carcinogenic in mice, rats, and hamsters, causing liver tumors in all three species and tumors of the spleen and thyroid in hamsters. There is equivocal evidence that hexachlorobenzene is teratogenic; reproductive effects have been observed in rats and monkeys. Humans accidentally exposed to hexachlorobenzene displayed numerous adverse effects, including enlarged livers, rheumatoid arthritis-like symptoms, and severe skin damage.

CAS Number: 118-74-1

Chemical Formula: C_6Cl_6

IUPAC Name: Hexachlorobenzene

Important Synonyms and Trade Names: HCB, perchlorobenzene

Chemical and Physical Properties

Molecular Weight: 285

Boiling Point: 326°C

Melting Point: 230°C

Specific Gravity: 1.57 at 20°C

Solubility in Water: 10 µg/liter at 25°C

Solubility in Organics: Soluble in acetone, ether, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 6.18

Vapor Pressure: 1×10^{-5} mm Hg at 20°C

Vapor Density: 918

Flash Point: 242°C

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Transport and Fate

Hexachlorobenzene (HCB) is persistent in the environment. Although it has a low vapor pressure, it may volatilize because of its low water solubility and high level of activity in water. HCB has a high log octanol/water partition coefficient and therefore would not be expected to move readily through soil. Also, its high specific gravity suggests that it would probably move through soil as a nonaqueous-phase liquid (NAPL) and not necessarily in the groundwater.

The major fate of hexachlorobenzene is probably nonpermanent sorption to organic material in the soil and sediment. Although this binding will immobilize HCB, it will not do so permanently, and desorption may produce continuous, low-level concentrations of HCB in the surrounding media. Organisms can bioaccumulate HCB, but it is unclear whether biomagnification occurs in the food chain. Degradation in the environment, occurs very slowly, if at all. The two possible routes of degradation are photolysis, possibly assisted by the presence of photosensitizing organic materials in aqueous media, and biodegradation by soil and aquatic organisms.

Health Effects

Hexachlorobenzene is carcinogenic in mice, rats, and hamsters. Liver tumors are induced in all three species. In addition, tumors of the spleen and thyroid were induced in HCB-treated hamsters (Cabral et al. 1977). There is equivocal evidence suggesting that HCB is teratogenic at high dose levels in rats (Khera 1974) and mice (Courtney et al. 1976). The addition of HCB to the diets of rats at 160 ppm (approximately 10 mg/kg/day) or more adversely affects reproduction (Grant et al. 1977). HCB has also had adverse effects on reproduction in monkeys (Iatropoulos et al. 1976). In an epidemic of HCB poisoning in Turkey in which the overall mortality rate among exposed persons was about 10%, 95% of the breast-fed infants whose mothers were exposed to HCB died. This incident was caused by consumption of seed grain that had been treated with a fungicide containing HCB; more than 3,000 people were affected by porphyria cutanea tarda, a defect in porphyrin metabolism caused by HCB. The affected individuals displayed severe skin manifestations including photosensitivity, increased pigmentation, bullae formation, deep scarring, a permanent increase in body hair, and atrophy of the skin. Many children were affected with rheumatoid arthritis-like symptoms, and about one-third of all victims had enlarged livers (Courtney 1979). A similar effect on porphyrin metabolism has been seen in experimental animals fed HCB. HCB also appears to have an adverse effect on the immune system in mice, and it is an inducer of mixed function oxidase enzymes in the liver.

Toxicity to Wildlife and Domestic Animals

Hexachlorobenzene was tested in several short-term aquatic bioassays, but no toxicity was observed at the limit of solubility of the compound. Quail fed 20 ppm or more of HCB in their diets for 90 days had increased liver weights, and the size and hatchability of their eggs decreased. Feeding Kestrels 20 or 80 ppm HCB caused histological damage to both their livers and kidneys. Field studies of predatory and specifically fish-eating birds showed some correlation between increased HCB levels and increased mortality, low breeding success, and increased porphyria. However, other contaminants could also have been responsible for these effects.

Reduced reproductive success was observed in mink fed 1, 5, or 25 ppm of HCB in their diets (Bleavins et al. 1984). Effects included decreased litter size, increased frequency of still births, increased fetal mortality, and decreased post-natal growth.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risk associated with lifetime exposure to various concentrations of hexachlorobenzene in water are:

Risk	Concentration
10^{-5}	7.2 ng/liter
10^{-6}	0.72 ng/liter
10^{-7}	0.07 ng/liter

CAG Unit Risk (USEPA): 1.67 $(mg/kg/day)^{-1}$

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HEXACHLOROBUTADIENE

Summary

Hexachlorobutadiene caused an increased incidence of kidney tumors in rats and was found to be mutagenic using the Ames assay. There is equivocal evidence that hexachlorobutadiene increases neonatal mortality. Chronic exposure to low levels of hexachlorobutadiene caused renal toxicity in rats and other studies have shown that exposure can affect the central nervous system and liver. Hexachlorobutadiene is also quite toxic to aquatic organisms.

CAS Number: 87-68-3

Chemical Formula: $Cl_2C{:}CClCCl{:}CCl_2$

IUPAC Name: Hexachloro-1,3-butadiene

Important Synonyms and Trade Names: Dolen, GP-40-66:120, HCBD, perchlorobutadiene, C46

Chemical and Physical Properties

Molecular Weight: 260.74

Boiling Point: 210 to 220°C

Melting Point: -19 to -22°C

Specific Gravity: 1.675 at 15.5°C

Solubility in Water: 2 mg/liter at 20°C

Solubility in Organics: Compatible with numerous resins; soluble in alcohol and ether

Log Octanol/Water Partition Coefficient: 4.8

Vapor Pressure: 0.15 mm Hg at 20°C

Transport and Fate

Hexachlorobutadiene (HCBD) is probably rather persistent in the environment. Volatilization and adsorption to organic particulates are apparently important transport processes for HCBD. In soil and sediments, HCBD is bound to organic material.

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This process acts as a sink for HCBD in the environment. There was no information on the ultimate fate of HCBD in nature in the sources searched.

Health Effects

The International Agency for Research on Cancer (IARC 1979) notes that there is limited evidence that hexachlorobutadiene is a carcinogen. Their conclusion is based on one oral feeding study in rats in which the incidence of kidney tumors increased in the animals of both sexes given the highest doses (Kociba et al. 1977). The results of a spot test of HCBD using the Ames assay were positive. The data on the reproductive toxicity of HCBD are equivocal. One study indicates that neonatal mortality rose following a single, subcutaneous injection of 20 mg/kg body weight to the dam just prior to mating. Another, more recent experiment exposed male and female rats to doses of 0.2, 2, and 20 mg/kg/day for 90 days prior to mating and 15 days during gestation; no toxic effects were noted in the offspring. However, male and female rats given 2 or 20 mg/kg/day of HCBD showed signs of renal toxicity. The results of a 2-year feeding study in rats confirmed that renal tubular hyperplasia was caused by doses larger than 2 mg/kg/day. Other studies have indicated that HCBD also affects the central nervous system and the liver (Harleman and Seinen 1979). HCBD is a cumulative toxin and is therefore more toxic after chronic exposures. The oral LD_{50} for adult rats is 250 mg/kg, and the LD_{50} for neonatal rats is one-quarter that for the adult animals.

Toxicity to Wildlife and Domestic Animals

Hexachlorobutadiene is very toxic to aquatic organisms, with 96-hour LC_{50} values for goldfish, rainbow trout, fathead minnow, and bluegill ranging from 90 to 330 µg/liter. Its chronic toxicity, as measured in an embryo-larval test in fathead minnows, is 9.3 µg/liter. Invertebrates and saltwater fish were affected at similar levels.

The ingestion of up to 30 ppm of HCBD in their diets (approximately 5-6 mg/kg) had no effect on Japanese quail.

No studies on the toxicity on HCBD to domestic animals were discussed in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of HCBD in water are:

Risk	Concentration
10^{-5}	4.5 μg/liter
10^{-6}	0.45 μg/liter
10^{-7}	0.045 μg/liter

CAG Unit Risk (USEPA): $7.75\text{x}10^{-2}$ $(\text{mg/kg/day})^{-1}$

ACGIH Threshold Limit Value: Suspected carcinogen 0.24 mg/m^3 TWA

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HEXACHLOROCYCLOHEXANE

Summary

Hexachlorocyclohexane (HCH) has four major isomers, alpha, beta, gamma, and delta, of which the gamma isomer (lindane) is generally the most active. HCH is fairly persistent in the environment. Three of the isomers caused liver tumors in mice when administered alone. Exposure to lindane decreased the number of live young produced by pregnant dogs. Lindane is also quite toxic to aquatic life.

Note: Information presented below should be considered generally applicable to the HCH isomers unless a specific isomer is indicated.

CAS Number: 608-73-1
alpha-HCH: 319-84-6
beta-HCH: 319-85-7
gamma-HCH: 58-89-9
delta-HCH: 319-86-8

Chemical Formula: $C_6H_6Cl_6$

IUPAC Name: 1,2,3,4,5,6-Hexachlorocyclohexane

Important Synonyms and Trade Names: Benzene hexachloride, HCH, Lindane (gamma-HCH), HCH

Chemical and Physical Properties

Molecular Weight: 290.82

Boiling Point: No available data

Melting Point: alpha-HCH: 158°C
beta-HCH: 310°C
gamma-HCH: 112°C
delta-HCH: 138°C
Technical HCH: 65°C

Solubility in Water: alpha-HCH: 10 mg/liter
beta-HCH: 5 mg/liter
gamma-HCH: 10 mg/liter
delta-HCH: 10 mg/liter
Technical HCH: 10-32 mg/liter

Log Octanol/Water Partition Coefficient: 3.8

Vapor Pressure: alpha-HCH: 2.5×10^{-5} mm Hg at 20°C
beta-HCH: 2.8×10^{-7} mm Hg at 20°C
gamma-HCH: 2×10^{-4} mm Hg at 20°C
delta-HCH: 1.7×10^{-5} mm Hg at 20°C

Transport and Fate

In general, the transport and fate of the hexachlorocyclohexane isomers is similar and they will therefore be discussed as a group. The primary transport and fate process for hexachlorocyclohexane in an aqueous system appears to be adsorption to organic particles, transport to anaerobic sediments, and subsequent biodegradation by anaerobic organisms. Volatilization may be somewhat important in the aquatic environment and is probably a major transport process in soils. It is important to note that biodegradation of hexachlorocyclohexane yields such chemicals as pentachlorocyclohexane, tetrachlorobenzene, and trichlorophenol and therefore may not result in substantial detoxification of the chemical. Lindane has been shown to be rather persistent when applied to soil, with up to 10 percent of an applied sample remaining after 10 years.

Health Effects

The alpha, beta, and gamma isomers of hexachlorocyclohexane have all been shown to cause liver tumors in mice but not in other tested species. HCH has not been thoroughly tested for genotoxic effects but does not appear to be mutagenic. The alpha, beta, and delta isomers have not been tested for their teratogenic or reproductive toxicological potential. Lindane has been tested and was not teratogenic, but in two studies it decreased the number of live young produced (Earl et al. 1973). Alpha-HCH has been shown to cause nonmalignant lesions in the liver of test animals at doses below those required to induce tumors. Lindane has been associated with the development of aplastic anemia in humans (West 1967).

Toxicity to Wildlife and Domestic Animals

Lindane (gamma-HCH) is responsible for the effectiveness of hexachlorocyclohexane as an insecticide and is generally more toxic than the other isomers or technical HCH. In fact, the presence of the other HCH isomers decreases the toxicity of lindane to aquatic organisms, either by an antagonistic effect or by decreasing the chemical's solubility. Therefore, the toxicity of lindane and HCH will be considered separately.

Lindane is acutely toxic to freshwater fish with LC_{50} values ranging from 2 μg/liter to 141 μg/liter; and to saltwater fish at levels of from 7.3 to 104 μg/liter. Lindane was acutely toxic to the pink shrimp at 0.17 μg/liter. Acute-chronic ratios for lindane ranged from 7.5 to 63, and therefore the Final Chronic Value for the protection of freshwater species was determined to be 0.08 μg/liter. Aquatic organisms appear to bioconcentrate between 100 and 500 times the steady-state concentration of lindane in the water.

Technical hexachlorocyclohexane was much less toxic than lindane, with acute toxicity ranging from 100 μg/liter to 15,000 μg/liter for freshwater fish. Data on saltwater species also indicated that the technical compound was less acutely toxic. No information was available on the chronic toxicity of HCH. A bioconcentration factor was not reported but is probably similar to that for lindane.

No studies on the toxicity of the HCH isomers to terrestrial or domestic animals was found in the literature reviewed. However, voles at Love Canal that had decreased lifespans and reproductive ability had high levels of lindane in their livers (Rowley et al. 1983).

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Hexachlorocyclohexane mixture:

The available data are inadequate for establishing final criteria for hexachlorocyclohexane mixture. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 100 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 0.34 μg/liter
Chronic toxicity: No available data

Lindane (gamma-HCH):

Freshwater

Acute toxicity: 2.0 μg/liter
Chronic toxicity: 0.08 μg/liter

Saltwater

Acute toxicity: 0.16 μg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of the HCH isomers in water are:

Risk	Alpha-HCH Concentration	Beta-HCH Concentration	Gamma-HCH Concentration	Technical HC Concentratio
10^{-5}	92 ng/liter	163 ng/liter	186 ng/liter	123 ng/liter
10^{-6}	9.2 ng/liter	16.3 ng/liter	18.6 ng/liter	12.3 ng/lite
10^{-7}	0.92 ng/liter	1.63 ng/liter	1.86 ng/liter	1.23 ng/lite

Interim Primary Drinking Water Regulation: gamma-HCH: 0.004 mg/lite

CAG Unit Risk (USEPA): alpha-HCH: 11.1 $(mg/kg/day)^{-1}$
beta-HCH: 1.84 $(mg/kg/day)^{-1}$
gamma-HCH: 1.33 $(mg/kg/day)^{-1}$
technical-HCH: 4.75 $(mg/kg/day)^{-1}$

OSHA Standard (air): gamma-HCH: 500 $\mu g/m^3$ TWA

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HEXACHLOROETHANE

Summary

Hexachloroethane produced liver tumors in mice when administered by gavage. It caused central nervous sytem effects, hepatic dysfunction; and renal damage at high doses in animal studies.

CAS Number: 67-72-1

Chemical Formula: C_2Cl_6

IUPAC Name: Hexachloroethane

Important Synonyms and Trade Names: Ethylene hexachloride, hexachloroethylene, carbon hexachloride

Chemical and Physical Properties

Molecular Weight: 237

Boiling Point: 187°C (sublimes)

Melting Point: 187°C (sublimes)

Specific Gravity: 2.09

Solubility in Water: 50 mg/liter

Solubility in Organics: Soluble in alcohol, benzene, chloroform, and ether

Log Octanol/Water Partition Coefficient: 3.34

Vapor Pressure: 0.4 mm Hg at 20°C

Transport and Fate

Hexachloroethane is relatively persistent in the environment. Volatilization may be an important transport process but probably occurs slowly from natural waters. Hexachloroethane's high log octanol/water partition coefficient suggests that it adsorbs to organics in the soil and sediment and that it may bioaccumulate. Biodegradation is unlikely to be a significant fate process.

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Health Effects

There is limited evidence that hexachloroethane is carcinogenic in experimental animals. In a National Cancer Institute study, hexachloroethane administered by gavage produced malignant liver tumors in male and female B6C3F1 mice. It did not cause a statistically significant increase in tumors in Osborne-Mendel rats, but some rare renal tumors did develop. Hexachloroethane has not been reported to be mutagenic. Reduced litter sizes were observed after oral administration of 5,500 mg/kg to pregnant rats.

Hexachloroethane's major physiological effect in animals is on the central nervous system. Oral doses of 1-1.4 g/kg caused weakness, staggering gait, and twitching muscles in dogs. Hepatic dysfunction and renal damage were also reported in various experiments. The oral LD_{50} for rats was 4,460 mg/kg. The dermal LD_{50} for rabbits was more than 32,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

Hexachloroethane is stored in animals' fat, and some bioaccumulation would be expected in animals higher on the food chain.

The 48-hour LC_{50} for *Daphia magna* is 8,070 μg/liter; the 48-hour LC_{50} for the larva of the midge, *Tanytarus dissimilis*, is 1,700 μg/liter. The 96-hour, static LC_{50} is 980 μg/liter for both the bluegill and the rainbow trout, and it is 2,400 μg/liter for the sheepshead minnow. In embryo-larval tests on the fathead minnow, the chronic toxicity value was reported to be 540 μg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of hexachloroethane in water are:

Risk	Concentration
10^{-5}	19 μg/liter
10^{-6}	1.9 μg/liter
10^{-7}	0.19 μg/liter

CAG Unit Risk (USEPA): 1.4×10^{-2} (mg/kg/day)$^{-1}$

OSHA Standard (skin): 10 mg/m^3 TWA

ACGIH Threshold Limit Value: 100 mg/m^3 TWA

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HEXACHLOROPHENE

Summary

Hexachlorophene severely damages neonates' central nervous systems. It has had teratogenic and reproductive effects in animals. There is suggestive evidence that hexachlorophene is also teratogenic in humans.

CAS Number: 70-30-4

Chemical Formula: $(C_6HCl_3OH)_2CH_2$

IUPAC Name: 2,2-Methylene-bis(3,4,6-trichlorophenol)

Important Synonyms and Trade Names: Hexide, Nabac

Chemical and Physical Properties

Molecular Weight: 406.9

Boiling Point: No available data

Melting Point: 165°C

Specific Gravity: No available data

Solubility in Water: Practically insoluble in water; estimated to be 50 mg/liter

Solubility in Organics: Soluble in acetone, alcohol, ether, and chloroform

Log Octanol/Water Partition Coefficient: 3.93

Vapor Pressure: Estimated to be 10^{-4} mm Hg at 20°C

Transport and Fate

No information on the transport and fate of hexachlorophene was found in the sources reviewed. Its water solubility and vapor pressure suggest that it would not be very volatile. Its high log octanol/water partition coefficient indicates that it is likely to be adsorbed to soil and sediments. Hexachlorophene is not degraded by laboratory animals. Therefore, bioaccumulation, and subsequent degradation, is unlikely to be an important fate. Hexachlorophene is probably persistent in the environment.

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Health Effects

In the 1960s, soap containing hexachlorophene was used to bathe neonates in hospital nurseries. Some of the infants developed symptoms of central nervous system damage, with twitching, convulsions, and death. The neurological damage, especially of the white matter of the cerebrum and brainstem, was severe. In animal studies, it has been shown that treatment with hexachlorophene inhibited the synthesis of myelin in the peripheral and central nervous systems.

Hexachlorophene was not carcinogenic in an NCI study in rats (IARC 1979). A series of in vivo and in vitro mutagenesis assays did not yield positive results. There is some evidence that a higher incidence of malformations among the children of hospital workers is due to repeated hexachlorophene exposure. Pregnant rats administered hexachlorophene in their diets or by gavage had smaller litters and an increased incidence of cleft palate and other fetal abnormalities. The oral LD_{50} of hexachlorophene is 60 mg/kg for both the rat and the guinea pig, and 67 mg/kg for the mouse. The dermal LD_{50} values are 1,840 mg/kg, 1,100 mg/kg, and 270 mg/kg, for the rat, guinea pig, and mouse, respectively.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of hexachlorophene to wildlife and domestic animals was available in the sources reviewed.

Regulations and Standards

No regulations or standards have been established for hexachlorophene.

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HEXANE

Summary

Fetotoxicity was produced by the administration of hexane to pregnant rats. In humans, hexane irritates the mucous membranes, eyes, and skin and can cause dermatitis and pulmonary edema. Chronic exposure to hexane can cause polyneuropathy with axonal degeneration of the peripheral nervous system.

CAS Number: 110-54-3

Chemical Formula: C_6H_{14}

IUPAC Name: n-Hexane

Important Synonyms and Trade Names: n-Hexane, hexyl hydride

Chemical and Physical Properties

Molecular Weight: 86.20

Boiling Point: 68.9°C

Melting Point: -94°C

Specific Gravity: 0.6603 at 20°C

Solubility in Water: 140 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol and ether

Log Octanol/Water Partition Coefficient: 4.3 (calculated)

Vapor Pressure: 124 mm Hg at 20°C

Vapor Density: 3.0

Flash Point: -21.7°C (closed cup)

Transport and Fate

Hexane volatilizes readily from surface water and reacts with OH radicals in the air. In the soil, it is partially adsorbed to the surface of organic materials but may leach into the groundwater.

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Health Effects

Hexane did not promote tumors when it was administered to mice topically and subcutaneously. No other data on the carcinogenicity of hexane were found (Kraemer et al. 1974). There were no mutagenicity data available in the literature surveyed.

Pregnant Fischer 344 rats were exposed to 1,000 ppm (3,600 mg/m^3) of hexane for 6 hours on the 20th day of gestation. Significant amounts of hexane and the neurotoxic metabolite 2,5-hexanedione appeared in the fetal tissues (Bus et al. undated). Fetotoxicity was observed in mice when pregnant females were given oral doses of 238 mg/kg from the 6th to 15th days of gestation. No evidence of teratogenicity was reported in the literature reviewed.

In humans, mild exposure to hexane vapors can irritate the mucous membranes. Exposure to air concentrations greater than 1% hexane may cause dizziness, unconsciousness, and death. Direct skin contact causes irritation and dermatitis. Inhalation exposure can cause chemical pneumonitus, pulmonary edema, and hemorrhage. Exposure to levels of 5,000 ppm (18,000 mg/m^3) for 10 minutes reportedly caused dizziness, and exposure to 5,400 mg/m^3 caused nausea, and eye and throat irritation.

Chronic exposure to hexane causes polyneuropathy with axonal degeneration of the distal parts of the peripheral nervous system, as well as the spinal cord and brain stem. Resulting afflictions are weakness, memory loss, numbness, and headaches. Yamamura (1969) described peripheral nervous system disturbances in 93 workers at a sandal manufacturing facility in Japan where hexane concentrations ranged from 1,800 to 9,000 mg/m^3. The oral LD_{50} for rats is reported to be 287 mg/kg.

A chronic oral study in which rats were given 400-600 mg/liter hexane in water per day (approximately 100-150 mg/kg/day) for 5 months resulted in peripheral neuropathy and distal nerve fiber degeneration. Repetitive subcutaneous injections or inhalation also produced peripheral neuropathy (Spenser and Schaumburg 1977).

Toxicity to Wildlife and Domestic Animals

No mortalities were reported among young Coho salmon exposed to 100 mg/liter hexane for 96 hours in sea water.

Regulations and Standards

OSHA Standard: 1,800 mg/m^3 TWA

ACGIH Threshold Limit Value: 180 mg/m^3 TWA (n-hexane)
1800 mg/m^3 TWA (other isomers)

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IRON

Summary

There is some evidence that high concentrations of certain soluble iron salts may be teratogenic. The ingestion of excess amounts of iron can irritate the gastrointestinal tract. Inhaling some iron-containing dusts and fumes can cause siderosis, a type of benign pneumoconiosis.

Background Information

Iron is the fourth most abundant element in the earth's crust. The pure metal is very reactive chemically. It corrodes readily in the presence of oxygen and moisture, forming iron (III) hydroxide [$Fe(OH)_3$].

CAS Number: 7439-89-6

Chemical Formula: Fe

Chemical and Physical Properties

Atomic Weight: 55.847

Boiling Point: 2,750°C

Melting Point: 1,535°C

Specific Gravity: 7.86

Solubility in Water: Insoluble

Solubility in Organics: Soluble in alcohol and ether

Transport and Fate

Elemental iron and many iron compounds, including $Fe(OH)_3$ and the iron oxides, are insoluble in water. Iron also tends to chelate with organic and inorganic matter. Consequently, much of the iron present in aquatic systems tends to partition into the bottom sediments. Iron has relatively low mobility in soil. Atmospheric transport of iron can occur.

Health Effects

Some studies have indicated that inhalation exposure to high concentrations of iron oxide is associated with increased risk of lung and laryngeal cancers in hematite miners and foundry workers. However, the significance of these findings is not established since exposures were to a mixture of substances, including radon gas and decomposition products of synthetic resins. Iron dextran solutions are reported to cause injection site sarcomas in experimental animals. Some iron compounds, notably ferrous sulfate, are reported to have high mutagenic activity in test systems. Intravenous injection of high concentrations of soluble iron salts is reported to cause teratogenic effects, including hydrocephalus and anophthalmia, in various species of experimental animals.

Iron is an essential element in plants and animals. However, the ingestion of excess amounts of iron produces toxic effects, primarily associated with gastrointestinal irritation. Severe poisoning may cause gastrointestinal bleeding, pneumonitis, convulsions, and hepatic toxicity. A dose of about 30 g of a soluble ferric salt is likely to be fatal in humans. Persons ingesting more than 30 mg/kg should be observed for clinical symptoms and possibly hospitalized. Chronic ingestion of excess iron may lead to hemosiderosis or hemochromatosis. Long-term inhalation exposure to iron-containing dusts and fumes, especially iron oxide, can produce siderosis. This condition is considered to be a type of benign pneumoconiosis that does not progress to fibrosis. Exposure to aerosols and mists of soluble iron salts may produce respiratory and skin irritation. The toxic effects of iron in experimental animals are similar to those observed in humans.

Toxicity to Wildlife and Domestic Animals

The available data are not adequate to characterize the toxicity of iron to wildlife or domestic animals. Iron is unlikely to cause ecological toxicity.

Regulations and Standards

OSHA Standard: 10 mg/m^3 TWA (iron oxide fume)

ACGIH Threshold Limit Values:

5 mg/m^3 TWA (iron oxide fume, as Fe)
10 mg/m^3 STEL (iron oxide fume, as Fe)
1 mg/m^3 TWA (soluble iron salts, as Fe)
2 mg/m^3 STEL (soluble iron salts, as Fe)

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ISOBUTYL ALCOHOL

Summary

Rats injected subcutaneously with isobutyl alcohol developed liver and gastrointestinal tumors. At high concentrations, it depresses the central nervous system and irritates the skin, eyes and throat in both animals and humans. High concentrations of isobutyl alcohol have also been shown to cause slight changes in the liver and kidneys of exposed mice.

CAS Number: 78-83-1

Chemical Formula: $CH_3CHCH_3CH_2OH$

IUPAC Name: 2-Methyl propanol

Important Synonyms and Trade Names: Isobutanol

Chemical and Physical Properties

Molecular Weight: 74

Boiling Point: 108°C

Melting Point: -108°C

Specific Gravity: 0.805 at 20°C

Solubility in Water: 95,000 mg/liter at 18°C

Solubility in Organics: Soluble in alcohol and ether

Log Octanol/Water Partition Coefficient: 1.0 (calculated)

Transport and Fate

No information on the transport and fate of isobutyl alcohol was found in the sources reviewed. However, likely transport and fate processes can be determined based on the general reactions of alcohols and the specific chemical and physical properties of this material.

Alcohols are very soluble in water and therefore probably are not very volatile, although some evaporation may occur. Oxidation is likely to be an important fate process in both surface water and the atmosphere. In soil, isobutyl alcohol is probably biodegraded by soil microorganisms.

Health Effects

Although the evidence on whether isobutyl alcohol is a tumorigenic agent when administered orally in rats is equivocal, this compound is carcinogenic when injected subcutaneously, producing liver and gastrointestinal tumors. Isobutyl alcohol reportedly causes mutations in *Escherichia coli* strain A and cytogenic effects in *Saccharomyces cerevisiae*. No reproductive or teratogenic effects have been reported.

Isobutyl alcohol at high concentrations depresses the central nervous system in both animals and man. Other symptoms of excessive exposure are irritation of the eye and throat, formation of vacuoles in the superficial layers of the cornea, and loss of appetite. Direct application of isobutyl alcohol irritates the skin, causing erythemia and hyperemia. A dose of 19,370 mg/m^3 inhaled for 136 hours has a narcotic effect in mice and causes slight changes in the liver and kidneys. The oral LD_{50} for rats is 2.46 g/kg, while the dermal LD_{50} for rabbits is 4.24 g/kg.

Toxicity to Wildlife and Domestic Animals

Limited information is available on the effects of isobutyl alcohol on the environment. Inhibition of cell division occurs at 280 mg/liter for the bacterium *Pseudomonas patida* and at 290 mg/liter for the alga *Microcystis aeruginosa*.

Regulations and Standards

OSHA Standard (air): 300 mg/m^3 TWA

ACGIH Threshold Limit Values: 150 mg/m^3 TWA
225 mg/m^3 STEL

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ISOPROPYL ETHER

Summary

Although similar to ethyl ether, isopropyl ether is considered to be more toxic and irritating. At high concentrations it causes narcosis and death.

CAS Number: 108-20-3

Chemical Formula: $(CH_3)_2CHOCH(CH_3)_2$

IUPAC Name: di-Isopropyl ether

Important Synonyms and Trade Names: 2-Isopropoxypropane, IPE, DIPE, di-isopropyloxide

Chemical and Physical Properties

Molecular Weight: 102.2

Boiling Point: 69°C

Melting Point: -60°C

Specific Gravity: 0.73 at 20°C

Solubility in Water: 2,000 mg/liter

Solubility in Organics: Miscible with alcohol and ethyl ether

Log Octanol/Water Partition Coefficient: Approximately 2.5 (calculated)

Vapor Pressure: 130 mm Hg at 20°C

Vapor Density: 3.52

Flash Point: -18°C (closed cup)

Transport and Fate

No specific information on the transport and fate of isopropyl ether was found in the sources reviewed. However, likely transport and fate mechanisms can be determined from information on chemical and physical properties and by inference from information on bis(2-chloroisopropyl)ether.

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The relatively high vapor pressure of isopropyl alcohol indicates that it probably volatilizes from surface water and soils. It is somewhat water soluble and therefore may leach through soil, although its log octanol/water partition coefficient of approximately 2.5 indicates that it may be adsorbed to soil organics. In one study, bis(2-chloroisopropyl)ether was collected 150 miles downstream from a point source at the levels one would expect based on calculations using only river dilution factors. This indicates that the ether is not volatilized, degraded, or sorbed to any great degree. The chemical is probably fairly persistent in the environment. Conversion of the ether to a peroxide probably occurs in the environment, but the actual rate is unclear.

Health Effects

No reports on carcinogenicity, mutagenicity, reproductive toxicity, or teratogenicity were found in the literature reviewed. Isopropyl ether is considered to be somewhat more toxic and irritating than ethyl ether. The only toxic effects exhibited were narcosis and death after the chemical was administered at high concentrations (narcosis occurred at about 42,000 mg/m^3). The oral LD_{50} in rats is 6,470 mg/kg.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity of isopropyl ether to wildlife or domestic animals are not available.

Regulations and Standards

OSHA Standard (air): 2,100 mg/m^3 TWA

ACGIH Threshold Limit Values: 1,050 mg/m^3 TWA
1,320 mg/m^3 STEL

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LEAD

Summary

Lead is a heavy metal that exists in one of three oxidation states, 0, +2, and +4. There is suggestive evidence that some lead salts are carcinogenic, inducing kidney tumors in mice and rats. Lead is also a reproductive hazard, and it can adversely affect the brain and central nervous system by causing encephalopathy and peripheral neuropathy. Chronic exposure to low levels of lead can cause subtle learning disabilities in children. Exposure to lead can also cause kidney damage and anemia, and it may have adverse effects on the immune system.

CAS Number: 7439-92-1

Chemical Formula: Pb

IUPAC Name: Lead

Chemical and Physical Properties

Atomic Weight: 207.19

Boiling Point: 1,740°C

Melting Point: 327.502°C

Specific Gravity: 11.35 at 20°C

Solubility in Water: Insoluble; some organic compounds are soluble

Solubility in Organics: Soluble in HNO_3 and hot, concentrated H_2SO_4

Transport and Fate

Some industrially produced lead compounds are readily soluble in water (USEPA 1979). However, metallic lead and the common lead minerals are insoluble in water. Natural compounds of lead are not usually mobile in normal surface or groundwater because the lead leached from ores is adsorbed by ferric hydroxide or combines with carbonate or sulfate ions to form insoluble compounds.

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Movement of lead and its inorganic and organolead compounds as particulates in the atmosphere is a major environmental transport process. Lead carried in the atmosphere can be removed by either wet or dry deposition. Although little evidence is available concerning the photolysis of lead compounds in natural waters, photolysis in the atmosphere occurs readily. These atmospheric processes are important in determining the form of lead entering aquatic and terrestrial systems.

The transport of lead in the aquatic environment is influenced by the speciation of the ion. Lead exists mainly as the divalent cation in most unpolluted waters and becomes adsorbed into particulate phases. However, in polluted waters organic complexation is most important. Volatilization of lead compounds probably is not important in most aquatic environments.

Sorption processes appear to exert a dominant effect on the distribution of lead in the environment. Adsorption to inorganic solids, organic materials, and hydrous iron and manganese oxides usually controls the mobility of lead and results in a strong partitioning of lead to the bed sediments in aquatic systems. The sorption mechanism most important in a particular system varies with geological setting, pH, Eh, availability of ligands, dissolved and particulate ion concentrations, salinity, and chemical composition. The equilibrium solubility of lead with carbonate, sulfate, and sulfide is low. Over most of the normal pH range, lead carbonate, and lead sulfate control solubility of lead in aerobic conditions, and lead sulfide and the metal control solubility in anaerobic conditions. Lead is strongly complexed to organic materials present in aquatic systems and soil. Lead in soil is not easily taken up by plants, and therefore its availability to terrestrial organisms is somewhat limited.

Bioaccumulation of lead has been demonstrated for a variety of organisms, and bioconcentration factors are within the range of 100-1,000. Microcosm studies indicate that lead is not biomagnified through the food chain. Biomethylation of lead by microorganisms can remobilize lead to the environment. The ultimate sink of lead is probably the deep oceans.

Health Effects

There is evidence that several lead salts are carcinogenic in mice or rats, causing tumors of the kidneys after either oral or parenteral administration. Data concerning the carcinogenicity of lead in hùmans are inconclusive. The available data are not sufficient to evaluate the carcinogenicity of organic lead compounds or metallic lead. There is equivocal evidence that exposure to lead causes genotoxicity in humans and animals. The available evidence indicates that lead presents a hazard to reproduction and exerts a toxic effect on conception, pregnancy, and the fetus in humans and experimental animals (USEPA 1977, 1980).

Many lead compounds are sufficiently soluble in body fluids to be toxic (USEPA 1977, 1980). Exposure of humans or experimental animals to lead can result in toxic effects in the brain and central nervous system, the peripheral nervous system, the kidneys, and the hematopoietic system. Chronic exposure to inorganic lead by ingestion or inhalation can cause lead encephalopathy, and severe cases can result in permanent brain damage. Lead poisoning may cause peripheral neuropathy in adults and children, and permanent learning disabilities that are clinically undetectable in children may be caused by exposure to relatively low levels. Short-term exposure to lead can cause reversible kidney damage, but prolonged exposure at high concentrations may result in progressive kidney damage and possibly kidney failure. Anemia, due to inhibition of hemoblobin synthesis and a reduction in the life span of circulating red blood cells, is an early manifestation of lead poisoning. Several studies with experimental animals suggest that lead may interfere with various aspects of the immune response.

Toxicity to Wildlife and Domestic Animals

Freshwater vertebrates and invertebrates are more sensitive to lead in soft water than in hard water (USEPA 1980, 1983). At a hardness of about 50 mg/liter $CaCO_3$, the median effect concentrations for nine families range from 140 μg/liter to 236,600 μg/liter. Chronic values for *Daphnia magna* and the rainbow trout are 12.26 and 83.08 μg/liter, respectively, at a hardness of about 50 mg/liter. Acute-chronic ratios calculated for three freshwater species ranged from 18 to 62. Bioconcentration factors, ranging from 42 for young brook trout to 1,700 for a snail, were reported. Freshwater algae show an inhibition of growth at concentrations above 500 μg/liter.

Acute values for twelve saltwater species range from 476 μg/liter for the common mussel to 27,000 μg/liter for the soft-shell clam. Chronic exposure to lead causes adverse effects in mysid shrimp at 37 μg/liter, but not at 17 μg/liter. The acute-chronic ratio for this species is 118. Reported bioconcentration factors range from 17.5 for the Quahog clam to 2,570 for the blue mussel. Saltwater algae are adversely affected at approximate lead concentrations as low as 15.8 μg/liter.

Although lead is known to occur in the tissue of many free-living wild animals, including birds, mammals, fishes, and invertebrates, reports of poisoning usually involve waterfowl. There is evidence that lead, at concentrations occasionally found near roadsides and smelters, can eliminate or reduce populations of bacteria and fungi on leaf surfaces and in soil. Many of these microorganisms play key roles in the decomposer food chain.

Cases of lead poisoning have been reported for a variety of domestic animals, including cattle, horses, dogs, and cats. Several types of anthropogenic sources are cited as the source of lead in these reports. Because of their curiosity and their indiscriminate eating habits, cattle experience the greatest incidence of lead toxicity among domestic animals.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed Criteria)

The concentrations below are for active lead, which is defined as the lead that passes through a 0.45-μm membrane filter after the sample is acidified to pH 4 with nitric acid.

Freshwater

Acute toxicity: $e^{(1.34\,[\ln(\text{hardness})]\,-\,2.014)}$ μg/liter

Chronic toxicity: $e^{(1.34\,[\ln(\text{hardness})]\,-\,5.245)}$ μg/liter

Saltwater

Acute toxicity: 220 μg/liter
Chronic toxicity: 8.6 μg/liter

Human Health

Criterion: 50 μg/liter

Primary Drinking Water Standard: 50 μg/liter

NIOSH Recommended Standard: 0.10 mg/m^3 TWA (inorganic lead)

OSHA Standard: 50 μg/m^3 TWA

ACGIH Threshold Limit Values:

0.15 mg/m^3 TWA (inorganic dusts and fumes)
0.45 mg/m^3 STEL (inorganic dusts and fumes)

Virtually no specific information on the toxicity of 2,4,5-T to wildlife or domestic animals is available. While 2,4,5-T is thought to have relatively low toxicity for vertebrate species, it has been reported that populations of invertebrates, including beneficial insect species, have been adversely affected at field concentrations. Invertebrates may be adversely affected both directly because of the compound's toxicity and indirectly because of the changes 2,4,5-T produces in vegetation growth patterns. Although 2,4,5-T is not reported to have large, direct toxic effects on livestock, there are reports of animal deaths due to alterations in plant chemistry and palatability after 2,4,5-T treatment.

Information on the effects of 2,4,5-T on aquatic species is also limited. Among fish, the LD_{50} value for perch is 55 mg/liter; for guppies, 8 mg/liter; and for rainbow trout, 1.3 mg/liter.

Regulations and Standards

OSHA Standard (air): 10 mg/m^3 TWA

ACGIH Threshold Limit Value: 10 mg/m^3 TWA

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LITHIUM

Summary

Lithium produced cleft palate in the offspring of pregnant rats and mice, and it caused other malformations in the rat fetuses. Exposure to various lithium compounds can cause eye, skin, and mucous membrane irritation; pulmonary emphysema; nausea; blurred vision; coma; and epileptic seizures.

Background Information

Lithium is a soft white metal that reacts exothermally with nitrogen at room temperature when the humidity is moderately high. It burns and explodes in contact with water, nitrogen, acids, and oxidizing agents.

CAS Number: 7439-93-2

Chemical Formula: Li

IUPAC Name: Lithium

Important Synonyms and Trade Names: Lithium metal

Chemical and Physical Properties

Molecular Weight: 6.9

Boiling Point: 1342°C

Melting Point: 180.5°C

Specific Gravity: 0.534 at 20°C

Solubility in Water: Decomposes in cold water

Solubility in Organics: Insoluble

Vapor Pressure: 1 mm Hg at 723°C

Flash Point: Not pertinent (combustible solid)

Transport and Fate

No specific information on the transport and fate of lithium was found in the literature reviewed. Lithium metal decomposes in water and forms soluble lithium salts. It moves readily

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with ground and surface water. The movement of lithium through soil is limited by the cation exchange potential of the soil.

Health Effects

There were no data on carcinogenic or mutagenic effects in the literature reviewed.

Rats were dosed intraperitonally with 50 mg/day on days 1, 4, 7, and 9 of gestation, followed by doses of 20 mg daily until day 17. Malformations were seen in the eye (62%) and external ear (45%); cleft palate also occurred (39%) (Wright et al. 1970). Cleft palate was also observed in mice (Szabo 1970).

The lethality of lithium chloride is dependent upon the Na^+ level in the body. Dogs survived an oral dose of 50 mg/kg for 150 days when they had normal Na^+ levels but died within 8 to 12 days if they had a low Na^+ level. The oral LD_{50} for lithium chloride in rats was determined to be 757 µg/kg. The LD_{50} in dogs for Li_2CO_3 was measured as 500 mg/kg. Lithium compounds cause nausea; vomiting; skin, eye, and lung irritation; and pulmonary emphysema in humans at doses as low as 7 mg/kg.

The chronic health effects of lithium are anorexia, fatigue, dehydration, diarrhea, vomiting, blurred vision, coma, and epileptic seizures. The target organ is the kidney, although lithium is distributed fairly evenly throughout the body.

Toxicity to Wildlife and Domestic Animals

There were no data available on the toxicity of lithium to wildlife and domestic animals.

Regulations and Standards

ACGIH Threshold Limit Value: 0.025 mg/m^3 TWA (lithium hydride)

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MAGNESIUM

Summary

Exposure to magnesium oxide fumes can cause metal fume fever in humans. Exposure to magnesium oxide dust can irritate the eyes and respiratory tract. Ingestion of very high levels of magnesium salts can cause central nervous sytem effects; it can also have a laxative action.

Background Information

Magnesium is the eighth most abundant element on earth. It is very reactive chemically and does not occur uncombined in nature. Finely divided magnesium can react with water to yield hydrogen gas and magnesium hydroxide. However, reaction of solid magnesium with water is self-limiting because of the formation of a film of magnesium hydroxide. As a result, elemental magnesium is considered insoluble in water.

CAS Number: 7439-95-4

Chemical Formula: Mg

IUPAC Name: Magnesium

Chemical and Physical Properties

Atomic Weight: 24.312

Boiling Point: 1107°C

Melting Point: 648.8°C

Specific Gravity: 1.738

Solubility in Water: Insoluble; most salts are very soluble

Transport and Fate

Most magnesium salts are very soluble at pH levels normally found in natural waters, and the magnesium ion is readily transported in surface water, soil, and groundwater. The extent of magnesium transport in soil is dependent, in part, on the cation exchange capacity of the soil. Evaporation of ocean spray particles and subsequent atmospheric transport of magnesium

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can occur. Atmospheric transport of dusts and fumes of compounds such as magnesim oxide can also occur.

Health Effects

There is no evidence to suggest that magnesium has carcinogenic, mutagenic, teratogenic, or reproductive effects in humans or experimental animals. Magnesium oxide fumes can produce metal fume fever in humans and experimental animals. Exposure to magnesium oxide dust may cause irritation of the eyes and respiratory tract. Human exposure to magnesium usually occurs by ingestion. Magnesium is an essential element for humans, animals, and plants. Ingestion of 3.6 to 4.2 mg/kg/day is thought to be adequate for maintenance of magnesium balance in humans. The average adult American is estimated to ingest 240 to 480 mg/kg/day in food and water. However, magnesium is absorbed relatively poorly by the gastrointestinal tract and also is readily excreted in the urine. Excessive magnesium retention in the body generally only occurs as a result of severe kidney disease. Symptoms of hypermagnesemia can include a sharp drop in blood pressure, and respiratory paralysis due to central nervous system depression. Ingestion of magnesium salts at concentrations over 700 mg/liter can have a laxative effect. However, humans can adapt to ingestion of these levels in a relatively short time. Magnesium has a very unpleasant taste in water at concentrations producing toxic effects.

Different magnesium compounds vary in the severity of their toxic effects to experimental animals. Such effects include central nervous system and purgative effects similar to those seen in humans. Subcutaneous injection of powdered magnesium or magnesium alloys can produce symptoms in experimental animals resembling gas gangrene. Application of powdered magnesium to abraded skin can produce an inflammatory reaction. However, these types of skin effects have not been reported in exposed workers.

Toxicity to Wildlife and Domestic Animals

Available data are not adequate to characterize the toxicity of magnesium to wildlife or domestic animals. Observed effects are generally related to deficiency symptoms.

Regulations and Standards

OSHA Standard: 15 mg/m^3 (magnesium oxide fume)

ACGIH Threshold Limit Values:

10 mg/m^3 TWA (magnesium oxide fume)
10 mg/m^3 TWA (magnesite, nuisance particulate)
20 mg/m^3 STEL (magnesite, nuisance particulate)

U.S. Department of Transportation: Flammable solid; dangerous when wet

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Summary

Manganese chloride produced lymphomas and manganese sulfate, tumors after injestion into mice. In humans, chronic exposure to manganese causes degenerative changes in the central nervous system in the form of a Parkinson-like disease; liver changes also occur. Acute exposure causes manganese pneumonitis.

CAS Number: 7439-96-5

Chemical Formula: Mn

IUPAC Name: Manganese

Chemical and Physical Properties

Atomic Weight: 54.938

Boiling Point: 1962°C

Melting Point: 1244°C

Specific Gravity: 7.20

Solubility in Water: Decomposes; some compounds are soluble

Transport and Fate

Manganese occurs most commonly in the +2 and +4 oxidation states in aquatic systems. Its solubility depends to a great extent on pH, dissolved oxygen, and presence of complexing agents. In saltwater, it is estimated that 85% or more of the manganese present exists in a soluble form. In freshwater, manganese can occur as the soluble ion, in complex organic ions, or in colloidal suspensions. Manganese often occurs at higher concentrations near the bottom of stratified lakes because it can be released from sediments, as the manganous ion, under reducing conditions.

In the soil, the concentration and chemical form in which manganese occur can be affected by pH, cation exchange capacity, drainage, organic matter content, and other factors. The solubility of manganese is increased at low pH and under reducing conditions. The presence of high concentrations of chlorides, nitrates, or sulfates may also increase solubility. Under these conditions, manganese is more easily taken up by plants

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or transported in aqueous solution. Lack of sufficient cation exchange sites, which are provided by organic matter or clay, can also result in greater leaching of manganese to surface or groundwater.

Atmospheric transport of manganese fumes or dusts can occur. These materials can be returned to the earth by wet or dry deposition.

Health Effects

There are no epidemiological studies suggesting that manganese or its compounds are carcinogenic or have teratogenic or reproductive effects in humans. Exposure to manganese chloride by intraperitoneal or subcutaneous routes was reported to cause lymphomas in mice. Manganese sulfate was found to produce tumors after intraperitoneal administration in mice. No other reports of unequivocal carcinogenic activity are available for common manganese compounds. Some manganese compounds, notably manganese chloride, have exhibited mutagenic activity in a variety of test systems. Manganese compounds do not appear to be teratogenic, however.

In humans, manganese dusts and compounds have relatively low oral and dermal toxicity, but they can cause a variety of toxic effects after inhalation exposure. Acute exposure to very high concentrations can cause manganese pneumonitis, increased susceptibility to respiratory disease, and pathologic changes including epithelial necrosis and mononuclear proliferation. Chronic manganese poisoning is more common, but generally occurs only among persons occupationally exposed to manganese compounds. Degenerative changes in the central nervous system are the major toxic effects. Early symptoms include emotional changes, followed by a masklike face, retropulsion or propulsion, and a Parkinson's-like syndrome. Liver changes are also frequently seen. Individuals with an iron deficiency may be more susceptible to chronic poisoning.

Duplication of human exposure symptoms in experimental animals has only been partially successful. In rabbits exposed by inhalation to manganese dust, manganese pneumonitis did not develop, but fibrotic changes in the lungs were observed. Central nervous system effects characteristic of chronic exposure in humans have only been reproduced in monkeys.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of the toxicity of manganese to wildlife or domestic animals are not available.

A 48-hour LC$_{50}$ value of 16 mg/liter of manganese is reported for embryos of the oyster *Crassostrea virginica*. For the softshell clam *Mya arenaria* a 168-hour LC$_{50}$ value of 300 mg/liter is reported.

Regulations and Standards

OSHA Standard: 5 mg/m^3 Ceiling Level

ACGIH Threshold Limit Values:

1 mg/m^3 TWA (fume)
3 mg/m^3 STEL (fume)
5 mg/m^3 Ceiling Level (dust and compounds)

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MERCURY

Summary

Both organic and inorganic forms of mercury are reported to be teratogenic and embryotoxic in experimental animals. In humans, prenatal exposure to methylmercury has been associated with brain damage. Other major target organs for organic mercury compounds in humans are the central and peripheral nervous system and the kidney. In animals, toxic effects also occur in the liver, heart, gonads, pancreas, and gastrointestinal tract. Inorganic mercury is generally less acutely toxic than organic mercury compounds, but it does affect the central nervous system adversely.

Background Information

Several forms of mercury, including insoluble elemental mercury, inorganic species, and organic species, can exist in the environment. In general, the mercurous (+1) salts are much less soluble than the more commonly found mercuric (+2) salts. Mercury also forms many stable organic complexes that are generally much more soluble in organic liquids than in water. The nature and solubility of the chemical species that occur in an environmental system depend on the redox potential and the pH of the environment.

CAS Number: 7439-97-6

Chemical Formula: Hg

IUPAC Name: Mercury

Chemical and Physical Properties (Metal)

Atomic Weight: 200.59

Boiling Point: 356.58°C

Melting Point: -38.87°C

Specific Gravity: 13.5939 at 20°C

Solubility in Water: 81.3 µg/liter at 30°C; some salts and organic compounds are soluble

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Solubility in Organics: Depends on chemical species

Vapor Pressure: 0.0012 mm Hg at 20°C

Transport and Fate

Mercury and certain of its compounds, including several inorganic species and dimethyl mercury, can volatilize to the atmosphere from aquatic and terrestrial sources. Volatilization is reduced by conversion of metallic mercury to complexed species and by deposition of HgS in reducing sediments, but even so atmospheric transport is the major environmental distribution pathway for mercury. Precipitation is the primary mechanism for removal of mercury from the atmosphere. Photolysis is important in the breakdown of airborne mercurials and may be important in some aquatic systems. Adsorption onto suspended and bed sediments is probably the most important process determining the fate of mercury in the aquatic environment. Sorption is strongest into organic materials. Mercury in soils is generally complexed to organic compounds.

Virtually any mercury compound can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance biomethylation include large amounts of available mercury, large numbers of bacteria, the absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic environments. Mercury is strongly bioaccumulated by numerous mechanisms. Methylmercury is the most readily accumulated and retained form of mercury in aquatic biota, and once it enters a biological system it is very difficult to eliminate.

Health Effects

When administered by intraperitoneal injection, metallic mercury produces implantation site sarcomas in rats. No other studies were found connecting mercury exposure with carcinogenic effects in animals or humans. Several mercury compounds exhibit a variety of genotoxic effects in eukaryotes. In general, organic mercury compounds are more toxic than inorganic compounds. Although brain damage due to prenatal exposure to methylmercury has occurred in human populations, no conclusive evidence is available to suggest that mercury causes anatomical defects in humans. Embryotoxicity and teratogenicity of methylmercury has been reported for a variety of experimental animals. Mercuric chloride is reported to be teratogenic in experimental animals. No conclusive results concerning the teratogenic effects of mercury vapor are available.

In humans, alkyl mercury compounds pass through the blood brain barrier and the placenta very rapidly, in contrast to inorganic mercury compounds. Major target organs are the central and peripheral nervous systems, and the kidney. Methylmercury is particularly hazardous because of the difficulty of eliminating it from the body. In experimental animals, organic mercury compounds can produce toxic effects in the gastrointestinal tract, pancreas, liver, heart, and gonads, with involvement of the endocrine, immunocompetent, and central nervous systems.

Elemental mercury is not highly toxic as an acute poison. However, inhalation of high concentrations of mercury vapor can cause pneumonitis, bronchitis, chest pains, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea. Soluble mercuric salts are highly poisonous on ingestion, with oral LD_{50} values of 20 to 60 mg/kg reported. Mercurous compounds are less toxic when administered orally. Acute exposure to mercury compounds at high concentrations causes a variety of gastrointestinal symptoms and severe anuria with uremia. Signs and symptoms associated with chronic exposure involve the central nervous system and include behavioral and neurological disturbances.

Toxicity to Wildlife and Domestic Animals

The toxicity of mercury compounds has been tested in a wide variety of aquatic organisms. Although methylmercury appears to be more toxic than inorganic mercuric salts, few acute or chronic toxicity tests have been conducted with it. Among freshwater species, the 96-hour LC_{50} values for inorganic mercuric salts range from 0.02 µg/liter for crayfish to 2,000 µg/liter for caddisfly larvae. Acute values for methylmercuric compounds and other mercury compounds are only available for fishes. In rainbow trout, methylmercuric chloride is about ten times more toxic to rainbow trout than mercuric chloride, which is acutely toxic at about 300 µg/liter at 10°C. Methylmercury is the most chronically toxic of the tested compounds, with chronic values for *Daphnia magna* and brook trout of 1.00 and 0.52 µg/liter, respectively. The acute-chronic ratio for *Daphnia magna* is 3.2.

Mean acute values for saltwater species range from 3.5 to 1,680 µg/liter. In general, molluscs and crustaceans are more sensitive than fish to the acute toxic effects of mercury. A life-cycle experiment with the mysid shrimp showed that inorganic mercury at a concentration of 1.6 µg/liter significantly influences time of appearance of first brood, time of first spawn, and productivity. The acute-chronic ratio for the mysid shrimp is 2.9.

Chronic dietary exposure of chickens to mercuric chloride at growth inhibitory levels causes immune suppression, with a differential reduction effect on specific immunoglobulins.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity: 1.1 μg/liter
Chronic toxicity: 0.20 μg/liter

Saltwater

Acute toxicity: 1.9 μg/liter
Chronic toxicity: 0.10 μg/liter

Human Health

Criterion: 144 ng/liter

Primary Drinking Water Standard: 0.002 mg/liter

NIOSH Recommended Standard: 0.05 mg/m^3 TWA (inorganic mercury)

OSHA Standard: 0.1 mg/m^3 Ceiling Level

ACGIH Threshold Limit Values:

0.01 mg/m^3 TWA (alkyl compounds)
0.03 mg/m^3 STEL (alkyl compounds)
0.05 mg/m^3 TWA (vapor)
0.1 mg/m^3 TWA (aryl and inorganic compounds)

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METHACRYLIC ACID
METHACRYLIC ACID, METHYL ESTER

Summary

Methacrylic acid caused DNA damage in *Escherichia coli*, and methyl methacrylate was found to be mutagenic using the Ames assay. Methyl methacrylate caused fetal deaths and developmental abnormalities in rats. Exposure to methacrylic acid in the air can cause eye irritation in humans.

CAS Number: Methacrylic acid: 79-41-4
Methacrylic acid, methyl ester: 80-62-6

Chemical Formula: Methacrylic acid: $C_2H_2CH_3COOH$
Methacrylic acid, methyl esther: $C_2H_2CH_3COOCH_3$

IUPAC Name: Methacrylic acid: 2-Methyl-2-propenoate
Methacrylic acid, methyl esther: Methyl-2-methyl-2-propenoate

Important Synonyms and Trade Names:

Methacrylic acid: Methacrylate
Methacrylic acid, methyl esther: Methyl methacrylate, Pegalan

Chemical and Physical Properties

Molecular Weight: Methacrylic acid: 86
Methacrylic acid, methyl esther: 100

Boiling Point: Methacrylic acid: 163°C
Methacrylic acid, methyl esther: 100°C

Melting Point: Methacrylic acid: 16°C
Methacrylic acid, methyl esther: -48°C

Specific Gravity: Methacrylic acid: 1.015 at 20°C
Methacrylic acid, methyl esther: 0.994 at 20°C

Solubility in Water: Slightly soluble in water

Solubility in Organics: Miscible in alcohol, ether, and acetone

Log Octanol/Water Partition Coefficient:

Methacrylic acid: 0.65 (calculated)
Methacrylic acid, methyl esther: 1.15 (calculated)

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Vapor Pressure: Methacrylic acid: 0.65 mm Hg at 20°C
Methacrylic acid, methyl esther: 40 mg/Hg at 25°C

Vapor Density: Methacrylic acid: 297

Flash Point: 77°C

Transport and Fate

The limited information available on the transport and fate of methacrylic acid (methacrylate) and methacrylic acid, methyl esther (methyl methacrylate) suggests that microbial biodegradation is an important fate process for both compounds. Methyl methacrylate is probably somewhat volatile and may also be hydrolyzed in a slightly acidic aqueous solution to methacrylate and methanol.

Health Effects

No data on the carcinogenic, embryotoxic, or teratogenic properties of methacrylic acid were found in the literature reviewed. It did cause DNA damage in *Escherichia coli*. Direct eye or skin contact with methacrylic acid can result in blindness or corrosion of the skin, but exposure to a saturated atmosphere of 3,500 mg/m^3 for 7 hours caused only eye irritation in rats. Rats exposed via inhalation to 1,050 mg/m^3 for 6 hours a day for 20 days suffered slight renal congestion. The dermal LD_{50} for rabbits is 500 to 1,000 mg/kg.

Methyl methacrylate was administered to male and female Wistar rats in their drinking water for two years, and no treatment-related tumors were observed. Nor were any tumors found after rats had methyl methacrylate applied to the back of their necks 3 times per week for 4 months; the rats were kept for the rest of their lives. Solid pieces of methyl methacrylate have caused sarcomas at the sites of implantation. Thus, the evidence on the carcinogenicity of methyl methacrylate is inconclusive. Methyl methacrylate was found to be mutagenic using the Ames assay without activation and to be clastogenic in rat bone marrow cells after inhalation exposure of 4 mg/m^3 for 16 weeks. There is evidence that inhalation exposure to sufficiently high levels of methyl methacrylate can cause fetal deaths and developmental abnormalities in rats. The rat oral LD_{50} is 7.87 g/kg; for the mouse, it is 5.2 g/kg.

Toxicity to Wildlife and Domestic Animals

Although no information was available in the literature reviewed on the environmental toxicity of methacrylic acid, limited information is available for methyl methacrylate. The TL_m (24-96 hour) values for the bluegill, fathead minnow, and guppy range from 159 to 500 mg/liter. The threshold for inhibition of all multiplication of the bacterium *Pseudomonas putida* is 100 mg/liter; cell mutiplication of the alga *Microsystic aeruginous* is inhibited at 120 mg/liter.

Regulations and Standards

OSHA Standard (air):

Methacrylic acid, methyl esther: 410 mg/m^3 TWA

ACGIH Threshold Limit Values:

Methacrylic acid: 70 mg/m^3 TWA
Methacrylic acid, methyl esther: 410 mg/m^3 TWA
510 mg/m^3 STEL

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METHANOL

Summary

Methanol is highly toxic to humans when ingested or inhaled as a vapor. It causes blindness, nausea, headaches, delirium, and death. In addition, inhalation of high doses had teratogenic effects on the cardiovascular and urogenital systems of pregnant rats.

CAS Number: 67-56-1

Chemical Formula: CH_3OH

IUPAC Name: Methanol

Important Synonyms and Trade Names: Methyl alcohol, wood alcohol

Chemical and Physical Properties

Molecular Weight: 32

Boiling Point: 64.5°C

Melting Point: -94°C

Specific Gravity: 0.791 at 20°C

Solubility in Water: Miscible in water

Solubility in Organics: Soluble in alcohol, acetone, ether, benzene, and chloroform

Log Octanol/Water Partition Coefficient: -0.97 (calculated)

Vapor Pressure: 96 mm Hg at 20°C

Vapor Density: 1.11

Flash Point: 12°C (closed cup)

Transport and Fate

No information on the transport and fate of methanol was found in the sources reviewed. However, based on the general reactions of alcohols and the specific chemical and physical properties of the material, likely transport and fate processes can be determined.

Alcohols are very soluble in water and therefore probably are not very volatile. Some evaporation is likely to occur, however, especially for a compound such as methanol with a relatively high vapor pressure. Oxidation is probably an important fate process in both surface water and the atmosphere. In soil, methanol is probably biodegraded by soil microorganisms.

Health Effects

No information on the carcinogenicity of methanol was found in the literature reviewed. Several studies suggest that methanol may have some mutagenic activity. High doses (26,000 mg/m^3) caused teratogenicity, including effects on the cardiovascular and urogenital systems, when administered to pregnant rats for 7 hours.

The toxic effects of drinking methanol, by mistaking it for ethyl alcohol, are well known. Ingestion of a few ounces of methanol may result in nausea; epigastric pain; vomiting; headaches; dizziness; delirium; visual disturbances, including blindness; and death (Treon 1963). Similar effects have also been reported after exposure to high levels of methanol vapor (Treon 1963). The characteristic blindness that may develop in exposed humans results from retinal destruction and degeneration of the optic nerve. Metabolites of methanol, particularly formaldehyde, are believed to be responsible for this effect (Cornish 1980). The metabolism of methanol in man also gives rise to formic acid, and this is partly responsible for the severe acidosis that develops in intoxicated individuals (Cornish 1980, Treon 1963). However, chronic exposure to low levels of methanol, is not expected to have serious adverse health effects.

Toxicity to Wildlife and Domestic Animals

Methanol has a 48-hour LC_{50} of 8,000 mg/liter in trout, and the 24-hour LD_0 and LD_{100} values in creek chub are 8,000 and 17,000 mg/liter, respectively. No effects were seen in Daphnia exposed to 10,000 mg/liter nor in a protozoa exposed to 1,250 mg/liter either. Algae were not affected at levels of less than 10,000 mg/liter either. The LC_{50} of a saltwater species, the brine shrimp, was 10,000 mg/liter.

No data were available on the effects of methanol on domestic animals or terrestrial wildlife in the literature reviewed.

Regulations and Standards

NIOSH Recommended Standards: 260 mg/m^3 TWA
1,040 mg/m^3 Ceiling Level

OSHA Standard (air): 260 mg/m^3 TWA

ACGIH Threshold Limit Values: 260 mg/m^3 TWA
325 mg/m^3 STEL

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METHYL CHLORIDE

Summary

Methyl chloride is carcinogenic in male mice, causing tumors of the kidney and liver. It was found to be mutagenic using the Ames assay. Methyl chloride has also been shown to be teratogenic; it produces heart defects in the offspring of exposed mice. Exposure to high concentrations adversely affects the central nervous system, kidney, and liver in humans.

CAS Number: 74-87-3

Chemical Formula: CH_3Cl

IUPAC Name: Chloromethane

Important Synonyms and Trade Names: Chloromethane, monochloromethane

Chemical and Physical Properties

Molecular Weight: 50.49

Boiling Point: -23.7°C

Melting Point: -97°C

Specific Gravity: 0.9159 at 20°C

Solubility in Water: 6,450 to 7,250 mg/liter at 20°C

Solubility in Organics: Miscible with chloroform, ether, and glacial acetic acid; soluble in alcohol

Log Octanol/Water Partition Coefficient: 0.91

Transport and Fate

Methyl chloride is a gas at normal environmental temperatures and therefore is unlikely to remain in soil or water. Experimental studies have found the half-life of methyl chloride in agitated water to be 27 minutes. Although this finding may not be directly applicable to natural waters, it does suggest rapid loss of the compound from water. Sorption of methyl chloride to soil or sediment has not been studied; however, its relatively low log octanol/water partition coefficient suggests that partition occurs primarily into air or water.

The major route of environmental degradation of methyl chloride is probably through oxidation in the troposphere. At this level of the atmosphere, the methyl chloride molecule is attacked by hydroxyl radicals via the mechanism of hydrogen abstraction. The primary product is formyl chloride.

Health Effects

Methyl chloride was found to be carcinogenic in male mice exposed to the compound via inhalation for a 2-year period. A significantly increased incidence of benign and malignant kidney tumors was found in animals exposed to 2,100 mg/m^3. An increased incidence of hepatocellular carcinomas that was marginally significant was also found using an actuarial analysis of the data. Negative results for carcinogencicity for female mice and male and female rats were obtained in the same study. Methyl chloride has been found to be mutagenic using the Ames assay, with and without a metabolic activating system. Methyl chloride has also been shown to be teratogenic in mice, causing heart defects in fetuses exposed in utero at an airborne concentration of 1,050 mg/m^3 on gestation days 6 to 17.

Methyl chloride is not considered to be highly toxic. Repeated or prolonged human exposure to sufficient concentrations (greater than 100 mg/m^3) can result in central nervous system (CNS) effects including blurred vision, headache, nausea, loss of coordination, and personality changes. Renal and hepatic toxicity have also been reported in humans. Animal studies show CNS effects and binding to sulfhydryl-containing cellular macromolecules. This latter effect interferes with metabolism and is probably responsible for the observed tissue toxicity.

Toxicity to Wildlife and Domestic Animals

The only information available on the effects of methyl chloride in wildlife is an acute study on the bluegill that reported an LC_{50} value of 500 mg/liter for this species. Data on the other chlorinated methanes indicate that aquatic toxicity declines with decreased chlorination. Thus methyl chloride should be less toxic than chloroform or carbon tetrachloride, neither of which had any effect on *Daphnia magna* or the fathead minnow, respectively, during chronic exposure to 3,400 µg/liter. No information on the toxicity of methyl chloride to terrestrial wildlife or domestic animals was found in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of halomethanes in water are:

Risk	Concentration
10^{-5}	1.9 μg/liter
10^{-6}	0.19 μg/liter
10^{-7}	0.019 μg/liter

OSHA Standards: 210 mg/m^3 TWA
420 mg/m^3 Ceiling Level

ACGIH Threshold Limit Values: 105 mg/m^3 TWA
205 mg/m^3 STEL

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METHYLENE CHLORIDE

Summary

Methylene chloride increased the incidence of lung and liver tumors and sarcomas in rats and mice. It was found to be mutagenic in bacterial test systems. In humans, methylene chloride irritates the eyes, mucous membranes, and skin. Exposure to high levels adversely affects the central and peripheral nervous systems and the heart. In experimental animals, methylene chloride is reported to cause kidney and liver damage, convulsions, and paresis.

CAS Number: 75-09-2

Chemical Formula: CH_2Cl_2

IUPAC Name: Dichloromethane

Important Synonyms and Trade Names: Methylene dichloride, methane dichloride

Chemical and Physical Properties

Molecular Weight: 84.93

Boiling Point: 40°C

Melting Point: -95.1°C

Specific Gravity: 1.3266 at 20°C

Solubility in Water: 13,200-20,000 mg/liter at 25°C

Solubility in Organics: Miscible with alcohol and ether

Log Octanol/Water Partition Coefficient: 1.25

Vapor Pressure: 362.4 mm Hg at 20°C

Vapor Density: 2.93

Transport and Fate

Volatilization to the atmosphere appears to be the major mechanism for removal of methylene chloride from aquatic systems and its primary environmental transport process (USEPA 1979). Photooxidation in the troposphere appears to be the dominant environmental fate of methylene chloride. Once in the troposphere, the compound is attacked by hydroxyl radicals, resulting in the formation of carbon dioxide, and to a lesser extent, carbon monoxide and phosgene. Phosgene is readily hydrolyzed to HCl and CO_2. About one percent of tropospheric methylene chloride would be expected to reach the stratosphere where it would probably undergo photodissociation resulting from interaction with high energy ultraviolet radiation. Aerial transport of methylene chloride is partly responsible for its relatively wide environmental distribution. Atmospheric methylene chloride may be returned to the earth in precipitation.

Photolysis, oxidation, and hydrolysis do not appear to be significant environmental fate processes for methylene chloride, and there is no evidence to suggest that either adsorption or bioaccumulation are important fate processes for this chemical. Although methylene chloride is potentially biodegradable, especially by acclimatized microorganisms, biodegradation probably only occurs at a very slow rate.

Health Effects

Methylene chloride is currently under review by the National Toxicology Program (NTP 1984, USEPA 1985). Preliminary results indicate that it produced an increased incidence of lung and liver tumors in mice and mammary tumors in female and male rats. In a chronic inhalation study, male rats exhibited an increased incidence of sarcomas in the ventral neck region (Burek et al. 1984). However, the authors suggested that the relevance and toxicological significance of this finding were uncertain in light of available toxicity data. Methylene chloride is reported to be mutagenic in bacterial test systems. It also has produced positive results in the Fischer rat embryo cell transformation test. However, it has been suggested that the observed cell-transforming capability may have been due to impurities in the test material. There is no conclusive evidence that methylene chloride can produce teratogenic effects.

In humans, direct contact with methylene chloride produces eye, respiratory passage, and skin irritation (USEPA 1985). Mild poisonings due to inhalation exposure produce somnolence, lassitude, numbness and tingling of the limbs, anorexia, and lightheadedness, followed by rapid and complete recovery. More severe poisonings generally involve correspondingly greater disturbances of the central and peripheral nervous systems. Methylene chloride also has acute toxic effects on the heart, including the induction of arrhythmia. Fatalities reportedly due to methylene chloride exposure have been attributed to cardiac injury and heart failure. Methylene chloride is metabolized to carbon monoxide in vivo, and levels of carboxyhemoglobin in the blood are elevated after acute exposures. In experimental animals, methylene chloride is reported to cause kidney and liver damage, convulsions, and distal paresis. An oral LD$_{50}$ value of 2,136 mg/kg, and an inhalation LC$_{50}$ value of 88,000 mg/m^3/30 min are reported for the rat.

Toxicity to Wildlife and Domestic Animals

Very little information concerning the toxicity of methylene chloride to domestic animals and wildlife exists (USEPA 1980). Acute values for the freshwater species *Daphnia magna*, the fathead minnow, and the bluegill are 224,000, 193,000, and 224,000 μg/liter, respectively. Acute values for the saltwater species, mysid shrimp and sheepshead minnow, are 256,000 and 331,000 μg/liter, respectively. No data concerning chronic toxicity are available. The 96-hour EC$_{50}$ values for both freshwater and saltwater algae are greater than the highest test concentration, 662,000 μg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 12.4 mg/liter (for protection against the noncarcinogenic effects of methylene chloride)

CAG Unit Risk (USEPA): 1.4×10^{-2} (mg/kg/day)$^{-1}$

NIOSH Recommended Standards:

261 mg/m^3 TWA in the presence of no more than 9.9 mg/m^3 of CO
1,737 mg/m^3/15 min Peak Concentration

OSHA Standards: 1,737 mg/m^3 TWA
3,474 mg/m^3 Ceiling Level
6,948 mg/m^3 Peak Concentration (5 min in any 3 hr)

ACGIH Threshold Limit Values: 350 mg/m^3 TWA
1,740 mg/m^3 STEL

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METHYL ETHYL KETONE

Summary

Methyl ethyl ketone (MEK) retarded fetal development and had some teratogenic effects in the offspring of exposed pregnant rats. At high doses, it affects the nervous system and irritates the eyes, mucous membranes, and skin. In addition, methyl ethyl ketone strongly potentiates the neurotoxic effects of n-hexane and n-hexamone.

CAS Number: 78-93-3

Chemical Formula: C_4H_8O

IUPAC Name: Butanone

Important Synonyms and Trade Names: Ethyl methyl ketone, MEK, 2-butanone

Chemical and Physical Properties

Molecular Weight: 72.1

Boiling Point: 79.6°C

Melting Point: -86.35°C

Specific Gravity: 0.805 at 20°C

Solubility in Water: Very soluble in water

Solubility in Organics: Miscible with alcohol, ether, benzene and acetone

Log Octanol/Water Partition Coefficient: 0.29

Vapor Pressure: 71.2 mm Hg at 20°C

Vapor Density: 2.41

Flash Point: 2°C

Transport and Fate

Very limited information on the transport and fate of methyl ethyl ketone was found in the literature reviewed. However, ketones in general are probably not very persistent.

Methyl ethyl ketone has a high vapor pressure and therefore would be expected to volatilize readily. However, because of its high water solubility, volatilization is probably limited in aquatic systems or wet soil. Once in the atmosphere it is apparently oxidized (Hoare and Whytock 1967). Methyl ethyl ketone has a low octanol water partition coefficient and therefore is probably not readily adsorbed. Biodegradation is probably the predominant fate of methyl ethyl ketone in the environment because of its aliphatic nature.

Health Effects

Methyl ethyl ketone has not been adequately tested for carcinogenicity and has produced only equivocal evidence of mutagenicity in a few bacterial assays. Schwetz et al. (1974) reported that MEK caused retarded fetal development and some teratogenic effects (acaudia, imperforate anus, and brachygnathia) at air concentrations of 3,000 ppm (approximately 9,000 mg/m^3). Methyl ethyl ketone is of relatively low toxicity but at high doses affects the nervous system and causes irritation of the eyes, nose, and skin. The oral LD$_{50}$ value for the rat was 2,750 mg/kg.

Although MEK is not strongly neurotoxic alone, it apparently strongly potentiates the neurotoxicity of n-hexane and n-hexanone (methyl n-isobutyl ketone).

Toxicity to Wildlife and Domestic Animals

Only limited information was available on the toxicity of methyl ethyl ketone to wildlife. LC$_{50}$ concentrations for two freshwater fishes were around 5,600 µg/liter (Turnball et al. 1954; Wallen et al. 1957). MEK was toxic to brine shrimp at LC$_{50}$ levels of 1950 mg/liter.

No information on the toxicity of MEK to terrestrial wildlife or domestic animals was found in the literature reviewed.

Regulations and Standards

NIOSH Recommended Standard: 590 mg/m^3 TWA

OSHA Standard (air): 200 ppm (590 mg/m^3) TWA

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METHYL ISOBUTYL KETONE

Summary

Methyl isobutyl ketone produced kidney damage in exposed rats. In humans, exposure has produced headaches, nausea, vomiting, and eye irritation.

CAS Number: 108-10-1

Chemical Formula: $(CH_3)_2CHCH_2COCH_3$

IUPAC Name: 4-Methyl-2-pentanone

Important Synonyms and Trade Names: Hexone, isobutyl methyl ketone, isopropyl acetone, MIK, and MIBK

Chemical and Physical Properties

Molecular Weight: 100.2

Boiling Point: 117°C

Melting Point: -84.7°C

Specific Gravity: 0.7978 at 20°C

Solubility in Water: Soluble

Solubility in Organics: Soluble in chloroform, alcohol, ether, acetone, benzene, and many other organic solvents

Log Octanol/Water Partition Coefficient: 1.18

Vapor Pressure: 16 mm Hg at 20°C

Vapor Density: 3.45

Flash Point: 23°C

Transport and Fate

Very limited information on the transport and fate of methyl isobutyl ketone (MIBK) was found in the literature reviewed. However, ketones in general are probably not very persistent. Methyl isobutyl ketone would be expected to volatilize fairly readily. However, because it is somewhat soluble in water, volatilization from wet environments is probably limited. Once in the atmosphere it is apparently oxidized. Methyl isobutyl ketone has a low octanol/water partition coefficient and therefore is probably not readily adsorbed. Biodegradation is probably the predominant fate of methyl isobutyl ketone in the environment. Evidence of this is provided by the biological oxygen demand value for methyl isobutyl ketone, which was 69% of the theoretical value after 20 days at 20°C.

Health Effects

No studies on the carcinogenicity, mutagenicity, reproductive toxicity or teratogenicity of methyl isobutyl ketone were found in the literature reviewed. Kidney damage was observed in rats exposed to 400 mg/m^3 of MIBK for 2 weeks but the damage appeared to be reversible. Methyl isobutyl ketone caused headache, nausea, vomiting, and eye irritation in a number of workers exposed to concentrations of 200 to 2,000 mg/m^3. The oral LD_{50} for MIBK in the rat was 2,080 mg/kg.

Toxicity to Wildlife and Domestic Animals

The only study on the toxicity of methyl isobutyl ketone to wildlife reported that the TL_{50} for brine shrimp was 1,230 mg/liter. MIBK is probably also not very toxic to other aquatic species or to terrestrial animals.

Regulations and Standards

NIOSH Recommended Standard: 200 mg/m^3 TWA

OSHA Standard (air): 400 mg/m^3 TWA

ACGIH Threshold Limit Values: 205 mg/m^3 TWA
300 mg/m^3 STEL

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METHYL PARATHION

Summary

Methyl parathion was embryotoxic, decreased reproductive potential, and lowered the survival rate of offspring when administered to experimental animals. Exposure also inhibits the activity of cholinesterase. Wild birds are particularly susceptible to the toxic effects of methyl parathion.

CAS Number: 298-00-0

Chemical Formula: $C_8H_{10}NO_5PS$

IUPAC Name: o,o-Dimethyl-o,p-Nitrophenylphosphorothioate

Important Synonyms and Trade Names: Metaphor, Wofatox

Chemical and Physical Properties

Molecular Weight: 263.23

Boiling Point: Thermally unstable; cannot be heated to normal boiling point

Melting Point: 37-38°C

Specific Gravity: 1.358 at 20°C

Solubility in Water: 50 mg/liter at 20°C

Solubility in Organics: Soluble in most organic solvents

Vapor Pressure: 9.7×10^{-4} mm Hg at 20°C

Transport and Fate

Methyl parathion is broken down quickly under environmental conditions, primarily by hydrolysis. Initial decomposition products in soil are p-nitrophenol and dimethylthiophosphoric acid. Methyl parathion will volatilize from soil and water. In the atmosphere, the sulfur atom is replaced by oxygen to yield methyl paraoxon, which is rapidly hydrolyzed. Bioaccumulation is probably not an important fate process for methyl parathion and although adsorption to soils may occur, it is also probably not an important fate process because of the rapid hydrolysis of the chemical. Methyl parathion is unlikely to leach through soil and enter the groundwater because of its low water solubility and short persistence.

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Health Effects

Methyl parathion was not carcinogenic when administered orally to rats and mice (NCI 1979), and was not mutagenic or only marginally positive for mutagenicity in numerous assays (Chen et al. 1981). Methyl parathion did induce sister chromatid exchange and caused cell cycle delay, however. It does not appear to be a teratogen but is a reproductive toxin causing decreased reproductive potential, decreased survival of offspring, and embryotoxicity.

Methyl parathion is converted in vivo to the oxygen analog, methyl paraoxon, which is responsible for its primary toxic effect: cholinesterase inhibition. Only one subchronic study was available on methyl parathion. This showed that continuous doses produced a steady decrease in enzyme activity but that upon cessation of dosing the animals recovered fairly quickly. Methyl parathion is quite acutely toxic, with an oral LD_{50} value in rats of about 15 mg/kg.

Toxicity to Wildlife and Domestic Animals

Methyl parathion was moderately toxic to freshwater and saltwater fish, with LC_{50} values ranging from 19,000 to 75,000 µg/liter. However, it was quite toxic to invertebrate species, with LC_{50} values between 2 and 50 µg/liter. The pesticide did not appear to affect rodent populations after field application but did cause some mortality in pheasant populations. Laboratory studies have also indicated that wild birds are quite susceptible to the toxic effects of methyl parathion; quail were more sensitive than pheasants or ducks. Methyl parathion was also quite toxic to nontarget insects. No reports of toxic effects on domestic animals were reported in the literature reviewed. Methyl parathion is fairly quickly metabolized in vivo to nontoxic products and is only slightly lipid soluble, and therefore is not expected to bioaccumulate or biomagnify in animals.

Regulations and Standards

NIOSH Recommended Standard (air): 0.2 mg/m^3 TWA

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NAPHTHALENE

Summary

Naphthalene retarded cranial ossification and heart development in the offspring of exposed pregnant rats. Inhalation exposure caused nausea, headache, and optic and kidney damage in humans and experimental animals. Oral administration produced cataracts in rabbits and induced changes in motor activity in rats and mice. Exposure to high doses of naphthalene cause severe hemolytic effects.

CAS Number: 91-20-3

Chemical Formula: $C_{10}H_8$

IUPAC Name: Naphthalene

Important Synonyms and Trade Names: Naphthene, tar camphor, moth balls

Chemical and Physical Properties

Molecular Weight: 128.16

Boiling Point: 217.9°C

Melting Point: 80.2°C

Specific Gravity: 1.152 at 20°C

Solubility in Water: 34.4 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, ether, acetone, and benzene

Log Octanol/Water Partition Coefficient: 3.37

Vapor Pressure: 0.087 mm Hg at 25°C

Vapor Density: 4.42

Transport and Fate

Environmental transport and fate is largely inferred from data for polycyclic aromatic hydrocarbons (PAHs) in general, because specific information for naphthalene is lacking. Rapid, direct photolysis of naphthalene to quinones may be an important

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process in surface waters. Oxidation is probably too slow to be a significant environmental process. However, data for some PAHs suggest that oxidation by chlorine or ozone may be a significant fate process when these oxidants are available insufficient quantity. Volatilization may play a role in transport depending on mixing rates in both the water column and air column. For naphthalene, adsorption is the most important aquatic transport process. Consideration of its log octanol/water partition coefficient and of the behavior of other PAHs indicate that naphthalene can be strongly adsorbed onto suspended and sedimentary particulate matter, especially particulates high in organic content. Dominance of volatilization or absorption as a transport process is directly related to environmental conditions. It is likely that this compound can be readily transported as adsorbed matter or suspended particulates in air or water.

Based on information concerning related compounds, it is likely that bioaccumulation of naphthalene is short term, especially for vertebrates. Although this compound is rapidly accumulated, it also is rapidly metabolized and excreted, and consequently bioaccumulation is not considered an important fate process. Naphthalene can be metabolized by multicellular organisms and degraded by microbes. Degradation by mammals is likely to be incomplete, with paten compound and the metabolites being excreted by the urinary system. Biodegradation by microorganisms is probably the ultimate fate process for naphthalene. Biodegradation generally appears to be more efficient in soil than in aquatic systems. However, experimental data indicate that biodegradation may be more important in those aquatic systems which are chronically affected by PAH contamination.

Atmospheric transport of PAHs can occur, and these materials can be returned to aquatic and terrestrial systems by wet and dry deposition. Some PAHs may enter surface and groundwaters by leaching from polluted soils.

Health Effects

There are no epidemiological or case studies available suggesting that naphthalene is carcinogenic in humans. This compound is not generally considered to be carcinogenic in experimental animals. However, there is equivocal evidence suggesting weak carcinogenic activity in rats after subcutaneous injection. Naphthalene is reported to produce DNA damage in mice after intraperitoneal injection. Retarded cranial ossification and heart development are reported among offspring of rats injected intraperitoneally with naphthalene on days 1 to 15 of gestation.

Little information concerning acute and chronic toxic effects is available. Inhalation exposure to naphthalene may cause headache, loss of appetite, nausea, and kidney damage in humans and experimental animals. Acute hemolytic effects are reportedly caused by ingestion or inhalation of relatively large quantities of naphthalene. Optical neuritis, injuries to the cornea, and opacities of the lens also may result after inhalation exposure or ingestion. Naphthalene is a mild eye irritant in rabbits, and cataracts can be induced after oral administration. Application to the skin produces erythema and slight edema in rabbits. Somnolence and changes in motor activity are observed after ingestion of naphthalene by rats and mice. Oral LD_{50} values of 1,250 mg/kg and 580 mg/kg are reported for the rat and the mouse, respectively.

Toxicity to Wildlife and Domestic Animals

The median effect concentrations for freshwater invertebrate species and three fish species are all reported to be greater than 2,300 μg/liter. Acute values reported for saltwater polychaete, oyster, and shrimp species are all greater than 2,350 μg/liter. A chronic value of 620 μg/liter and an acute-chronic ratio of 11 is reported for the fathead minnow, a freshwater species. No chronic values are available for saltwater species. Freshwater algae appear to be less sensitive to the effects of naphthalene than animal species. No information concerning saltwater plant species is available. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 10.5.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data are not adequate for establishing criteria.

OSHA Standard: 50 mg/m^3 TWA

ACGIH Threshold Limit Values: 50 mg/m^3 TWA
75 mg/m^3 STEL

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NICKEL

Summary

In a number of epidemiological studies, occupational exposure to nickel compounds has been associated with excess cancer of the lung and nasal cavity. In addition, inhalation exposure to nickel subsulfide and nickel carbonyl has been shown to cause cancer in rats, while studies of other nickel compounds administered to animals by other routes have reported carcinogenic effects as well. Several nickel compounds are mutagenic and can cause cell transformation. In humans, nickel and nickel compounds can cause a sensitization dermatitis. The chronic toxicity of nickel to aquatic organisms is high.

Background Information

The commonly occurring valences of nickel are 0, +1, +2, and +3, with +4 rarely encountered. Although elemental nickel is seldom found in nature and is not soluble in water, many nickel compounds are highly soluble in water. Nickel is almost always found in the divalent oxidation state in aquatic systems.

CAS Number: 7440-02-0

Chemical Formula: Ni

IUPAC Name: Nickel

Chemical and Physical Properties

Atomic Weight: 58.71

Boiling Point: 2,732°C

Melting Point: 1,453°C

Specific Gravity: 8.902 at 25°C

Solubility in Water: Insoluble; some salts are soluble

Solubility in Organics: Depends on the properties of the specific nickel salt

Vapor Pressure: 1 mm Hg at 1,810°C

Transport and Fate

Nickel is a highly mobile metal in aquatic systems because many nickel compounds are highly soluble in water. However, the insoluble sulfide is formed under reducing conditions and in the presence of sulfur. Above pH 9, precipitation of the hydroxide or carbonate exhibits some control on nickel mobility. In aerobic environments below pH 9, soluble compounds are formed with hydroxide, carbonate, sulfate, and organic ligands.

In natural, unpolluted waters, sorption and coprecipitation processes involving hydrous iron and manganese oxides are probably at least moderately effective in limiting the mobility of nickel. In more organic-rich, polluted waters, it appears that little sorption of nickel is likely. The lack of other controls on nickel mobility probably makes incorporation into bed sediments an important fate of nickel in surface waters. However, much of the nickel entering the aquatic environment will be transported to the oceans.

In general, nickel is not accumulated in significant amounts by aquatic organisms. Bioconcentration factors are usually on the order of 100 to 1,000. Uptake of nickel from the soil by plants can also occur. Photolysis, volatilization, and biotransformation are not important environmental fate processes for nickel. However, atmospheric transport of nickel and nickel compounds on particulate matter can occur.

Health Effects

There is extensive epidemiological evidence indicating excess cancer of the lung and nasal cavity for workers at nickel refineries and smelters, and weaker evidence for excess risk in workers at nickel electroplating and polishing operations. Respiratory tract cancers have occurred in excess at industrial facilities that are metallurgically diverse in their operations. The nickel compounds that have been implicated as having carcinogenic potential are insoluble dusts of nickel subsulfide and nickel oxides, the vapor of nickel carbonyl, and soluble aerosols of nickel sulfate, nitrate, or chloride. Inhalation studies with experimental animals suggest that nickel subsulfide and nickel carbonyl are carcinogenic in rats. Evidence for the carcinogenicity of nickel metal and other compounds is relatively weak or inconclusive. Studies with experimental animals indicate that nickel compounds can also produce various types of malignant tumors in experimental animals after administration by other routes, including subcutaneous, intramuscular, implantation, intravenous, intrarenal, and intrapleural. Carcinogenic potential is not strongly dependent on route or site of administration but appears to be inversely related to the solubility of the compounds in aqueous media. Insoluble compounds, such as nickel dust, nickel sulfide, nickel carbonate, nickel oxide, nickel carbonyl, and nickelocene are carcinogenic, whereas soluble nickel salts such as nickel chloride, nickel sulfate, and nickel ammonium sulfate, are not.

Mammalian cell transformation data indicate that several nickel compounds are mutagenic and can cause chromosomal alterations. The available information is inadequate for assessing teratogenic and reproductive effects of nickel in humans and experimental animals.

Dermatitis and other dermatological effects are the most frequent effects of exposure to nickel and nickel-containing compounds. The dermatitis is a sensitization reaction. Most information regarding acute toxicity of nickel involves inhalation exposure to nickel carbonyl. Clinical manifestations of acute poisoning include both immediate and delayed symptoms. Acute chemical pneumonitis is produced, and death may occur at exposures of 30 ppm (107 mg/m^3) for 30 minutes. Rhinitis, nasal sinusitis, and nasal mucosal injury are among the effects reported among workers chronically exposed to various nickel compounds. Studies with experimental animals suggest that nickel and nickel compounds have relatively low acute and chronic oral toxicity.

Toxicity to Wildlife and Domestic Animals

In freshwater, toxicity depends on hardness; nickel tends to be more toxic in softer water. Acute values for exposure to a variety of nickel salts, expressed as nickel, range from 510 μg/liter for *Daphnia magna* to 46,200 μg/liter for banded killifish at comparable hardness levels. Chronic values range from 14.8 μg/liter for *Daphnia magna* in soft water to 530 μg/liter for the fathead minnow in hard water. Acute-chronic ratios for *Daphnia magna* range from 14 in hard water to 83 in soft water, and are approximately 50 in both hard and soft water for the fathead minnow. Residue data for the fathead minnow indicate a bioconcentration factor of 61. Freshwater algae experience reduced growth at nickel concentrations as low as 100 μg/liter.

Acute values for saltwater species range from 152 μg/liter for mysid shrimp to 350,000 μg/liter for the mummichog. A chronic value of 92.7 μg/liter is reported for the mysid shrimp, which gives an acute-chronic ratio of 5.5 for the species. Reduced growth is seen in saltwater algae at concentrations as low as 1,000 μg/liter. Bioconcentration factors ranging from 299 to 416 have been reported for the oyster and mussel.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(0.76\,[\ln(\text{hardness})] + 4.02)}$ μg/liter

Chronic toxicity: $e^{(0.76\,[\ln(\text{hardness})] + 1.06)}$ μg/liter

Saltwater

Acute toxicity: 140 μg/liter
Chronic toxicity: 7.1 μg/liter

Human Health

Criterion: 13.4 μg/liter

CAG Unit Risk (USEPA): 1.15 $(\text{mg/kg/day})^{-1}$

NIOSH Recommended Standard: 15 μg/m^3 TWA (inorganic nickel)

OSHA Standard: 1 mg/m^3 (metal and soluble compounds, as nickel)

ACGIH Threshold Limit Values:

0.1 mg/m^3 TWA (soluble compounds, as nickel)
0.3 mg/m^3 STEL (soluble compounds, as nickel)
0.35 mg/m^3 TWA (nickel carbonyl, as nickel)
1 mg/m^3 TWA (nickel sulfide roasting, fume and dust, as nickel; human carcinogen)

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NITROCELLULOSE

Summary

Nitrocellulose with a large percentage of the high-nitrogen form is explosive.

Background Information

Nitrocellulose generally consists of a mixture of high-nitrogen and low-nitrogen (pyroxylin) forms of nitrated cellulose. The ratio of the two forms in a particular nitrocellulose compound varies. Nitrocellulose with a large amount of the high-nitrogen form is explosive, while nitrocellulose with mostly the low-nitrogen form is more stable.

CAS Number: 9004-70-0

Chemical Formula: $C_6H_7O_2(ONO_2)_x$

IUPAC Name: Cellulose nitrate

Important Synonyms and Trade Names: Cellulose tetranitrate, nitro cotton, soluble gun cotton, collodion

Chemical and Physical Properties

Molecular Weight: >504

Boiling Point: Explosive solid

Melting Point: 671°C

Specific Gravity: 1.35-1.6

Solubility in Water: Insoluble

Solubility in Organics: Soluble in ether and alcohol

Flash Point: 13°C

Transport and Fate

The limited information on the transport and fate of nitrocellulose indicates that it emits high levels of nitrate and nitrite when present in a landfill. This suggests that nitrocellulose may eventually degrade to cellulose or at least to fairly stable low-nitrogen nitrocellulose. After prolonged storage, nitrocellulose plastics emit camphor. This decay

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can decrease decomposition temperature and thus speed up decomposition, probably with the formation of nitrates and nitrites.

Health Effects

According to the extremely limited information on the toxicity of nitrocellulose, it is not very toxic. Its explosive tendency would be the primary concern associated with exposure to nitrocellulose at a waste site.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of nitrocellulose to wildlife and domestic animals was found in the sources reviewed.

Regulations and Standards

No regulations or standards based on the toxicity of nitrocellulose have been established.

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NITROPHENOL

Summary

Nitrophenol is reported to cause liver and kidney damage in experimental animals. It may have a direct effect on cell membranes in general.

CAS Number: 2-nitrophenol: 88-75-5
3-nitrophenol: 554-84-7
4-nitrophenol: 100-02-7

Chemical Formula: $C_6NO_2H_4OH$

IUPAC Name: o-, m-, or p-Nitrophenol

Important Synonyms and Trade Names: Nitrophenol, hydroxynitrobenzene, mononitrophenol

Chemical and Physical Properties

Molecular Weight: 139.11

Boiling Point: 2-nitrophenol: 216°C
3- and 4-nitrophenol: 279°C

Melting Point: 2-nitrophenol: 45.3°C
3-nitrophenol: 97°C
4-nitrophenol: 113°C

Specific Gravity: 1.5 at 20°C

Solubility in Water: 2,100 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, acetone, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 1.76

Vapor Pressure: 1 mm Hg at 50°C

pKa: 7.2

Transport and Fate

Based on information concerning 4-nitrophenol, it appears that photooxidation of the nitrophenols to catechol and nitrohydroquinone is their primary degradative pathway. There is, however, a possibility that organic material to which nitrophenol becomes adsorbed may act as a reducing agent in the photoreduction of this compound to aminophenol and dihydroxyazobenzene. Oxidation by hydroxyl radicals may also occur. Consideration of the low vapor pressure, relative high solubility in water, and moderate ionization constant of nitrophenol suggests that volatilization is not an important transport process. Although sorption of nitrophenol by organic materials probably occurs to only a limited extent, it appears that stable complexes with clay mineral and soils can be formed. Furthermore, there is a small possibility that nitrophenol can undergo hydrolysis within the clay structure.

Bioaccumulation and biomagnification do not appear to be important processes for nitrophenol. Biotransformation processes, including reduction of the nitro group, hydroxylation of the aromatic ring, and displacement of the nitro group by a hydroxy group, can be demonstrated with soil or water microorganisms under optimal conditions. Some studies suggest that nitrophenol is very persistent in aqueous soil cultures and can inhibit microbial growth in natural aquatic systems through its action as an oxidative phosphorylation uncoupler. However, results of other studies suggest that it is readily and rapidly degraded, especially by acclimated microorganism populations.

Health Effects

Based on the results of limited testing, nitrophenol does not appear to pose carcinogenic or mutagenic hazards (USEPA 1980). 4-Nitrophenol is currently being tested for carcinogenicity by the National Toxicology Program. No data concerning teratogenic potential are available.

Very little information concerning the toxicity of nitrophenol is available. This compound is reported to cause kidney and liver injury in experimental animals. Administration of 10 mg of 4-nitrophenol, 30 mg of 3-nitrophenol or 100 mg of 2-nitrophenol by gavage to anesthetized rats is reported to significantly increase respiratory volume. Nitrophenol can inhibit chlorine transport in red blood cells, suggesting a direct effect on cell membranes.

A significant increase in blood platelet levels was observed in rats after intraperitoneal injection of as little as 0.1 mg/kg of 2-nitrophenol. This effect was not seen when the other nitrophenols were administered. Oral LD$_{50}$ values of 620 mg/kg for 4-nitrophenol, 930 mg/kg for 3-nitrophenol and 2,828 mg/kg for 2-nitrophenol are reported for the rat.

Toxicity to Wildlife and Domestic Animals

Reported 24-hour LC_{50} values range from 35,000 μg/liter for 4-nitrophenol to 210,000 μg/liter for 2-nitrophenol for the freshwater species Daphnia magna and from 8,000 (4-nitrophenol) to 67,000 (2-nitrophenol) μg/liter for the bluegill. A concentration of 33,300 μg/liter of 2-nitrophenol caused 38% mortality in goldfish in eight hours. 96-Hour lethal threshold values of 26,000 (4-nitrophenol) and 32,900 μg/liter 2-nitrophenol are reported for the saltwater shrimp, Crangon septemspinosa.

A nitrophenol concentration of 35,000 μg/liter inhibits chlorophyll synthesis after 3 days in the freshwater alga, Chlorella pyrenoidosa. Growth of duckweed is reduced 50% by a concentration of 62,550 μg/liter of 2-nitrophenol.

The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 2.33.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data are not adequate for establishing criteria.

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WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2332 pages

PENTACHLOROPHENOL

Summary

Pentachlorophenol is probably persistent in natural environments. It is embryotoxic and fetotoxic. Chronic exposure has been shown to cause chloracne, headache, muscle weakness, weight loss, and liver and kidney damage. Technical grade pentachlorophenol is often contaminated with polychlorinated dibenzo-p-dioxins, and these contaminants may be responsible for some of the toxic effects associated with exposure to pentachlorophenol. Pentachlorophenol is highly toxic to aquatic organisms.

CAS Number: 87-86-5

Chemical Formula: C_6Cl_5OH

IUPAC Name: 2,3,4,5,6-Pentachlorophenol

Important Synonyms and Trade Names: PCP, DP-2 antimicrobial, Dowicide 7, Durotox.

Chemical and Physical Properties

Molecular Weight: 266.32

Boiling Point: Decomposes at 309°C

Melting Point: 190-191°C

Specific Gravity: 1.978 at 20°C

Solubility in Water: 14 mg/liter at 20°C; the sodium salt of pentachlorophenate is highly soluble in water

Solubility in Organics: Very soluble in alcohol and ether; soluble in hot benzene; slightly soluble in ligroin and other solvents

Log Octanol/Water Partition Coefficient: 5.01

Vapor Pressure: 1.1×10^{-4} mm Hg at 20°C

pKa: 4.74

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Transport and Fate

There is little information on the transport of pentachlorophenol through the environment. The compound has a low vapor pressure and, therefore, is not likely to volatilize readily. It is slightly soluble in water and does adsorb to sediments, and therefore may be transported through soil, surface water, and groundwater.

Pentachlorophenol is degraded by sunlight to lower chlorinated phenols, tetrachlorodihydroxyl benzenes, and non-aromatic fragments. The importance of photodegradation of pentachlorophenol in the environment is unknown. Soil microorganisms have also been found to degrade pentachlorophenol. However, the compound was persistent in sediments and leaf litter following a spill into a freshwater lake. Limited information on bioconcentration of pentachlorophenol in freshwater species suggest a bioconcentration factor of 500; in saltwater species factors vary from 13 to 3,830. Some pentachlorophenol residues found in tissue may actually be the result of metabolism of hexachlorobenzene.

Health Effects

Pentachlorophenol has not been found to be mutagenic or carcinogenic in the studies reviewed. It is currently under study by the National Toxicology Program for its carcinogenic potential. No teratogenic effects have been reported in the studies reviewed, but pentachlorophenol has been shown to be embryotoxic and fetotoxic. Pentachlorophenol has not been found to be highly toxic upon chronic exposure, although fatal cases from acute and chronic human exposures have been reported. Chloracne is the major effect associated with human chronic exposures, however, this may actually be caused by the polychlorinated dibenzo-dioxin contaminants found in technical grade pentachlorophenol. Other effects associated with chronic intoxication include muscle weakness, headache, anorexia, abdominal pain, weight loss, and effects on the liver and kidneys. Effects on the liver and kidney were less severe in animals treated with purified pentachlorophenol compared to technical grade compound.

Toxicity to Wildlife and Domestic Animals

Concentrations ranging from 34 to 2,000 μg/liter have been found to be acutely toxic to freshwater aquatic organisms. Toxicity is greater at acidic pH values than alkaline pH values. Growth of salmonid fish species is affected by pentachlorophenol at even lower concentrations. Some freshwater aquatic plants have also been shown to be sensitive to the compound but this has not been studied in detail.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Health criterion: 1.01 mg/liter
Organoleptic criterion: 30 μg/liter

OSHA Standard: 500 $\mu g/m^3$ TWA

ACGIH Threshold Limit Values: 0.5 mg/m^3 TWA
1.5 mg/m^3 STEL

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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RAO, K.R., ed. 1978. Pentachlorophenol: Chemistry, Pharmacology, and Environmental Toxicology. Environmental Science Research Volume 12. Plenum Press, New York

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Vol. 2. Washington, D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Pentachlorophenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-065

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Pentachlorophenol. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H043 (Final Draft)

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

PHENANTHRENE

Summary

Phenanthrene is a polycyclic aromatic hydrocarbon (PAH) and is moderately persistent in natural environments. In two skin painting studies, it produced application-site tumors, and it was shown to be mutagenic in several other studies. Workers exposed to materials containing phenanthrene developed chronic dermatitis and other skin disorders.

CAS Number: 85-01-8

Chemical Formula: $C_{14}H_{10}$

IUPAC Name: Phenanthrene

Chemical and Physical Properties

Molecular Weight: 178.24

Boiling Point: 340°C

Melting Point: 101°C

Specific Gravity: 1.025

Solubility in Water: 1.29 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, ether, acetone, benzene, and acetic acid

Log Octanol/Water Partition Coefficient: 4.46

Vapor Pressure: 6.8×10^{-4} mm Hg at 20°C

Vapor Density: 6.14

Transport and Fate

Much of the information concerning transport and fate is inferred from data for polycyclic aromatic hydrocarbons (PAHs) in general because specific information for phenanthrene is lacking. Rapid, direct photolysis of phenanthrene to quinones may occur in aqueous solution. Oxidation is probably too slow to be a significant environmental process and the available data suggest that volatilization generally is not an important transport process. The calculated log octanol/water partition

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coefficient of 4.46 indicates that the compound should be strongly absorbed onto particulate matter, especially particulates high in organic content. It is likely that phenanthrene can be transported as absorbed matter on suspended particulates in air or water. Data for PAHs in general indicate that phenanthrene will accumulate in the sediment and biota of the aquatic environment. Removal rates associated with absorption and subsequent sedimentation are probably slower than photolysis and degradation, but may be competitive with volatilization.

Data for a variety of PAHs suggest that bioaccumulation is a short term process, and long-term partitioning into biota is not a significant fate process. Phenanthrene can be metabolized by multicellular organisms and degraded by microbes.

Degradation by mammals is likely to be incomplete, with parent compound and the metabolites being excreted by the urinary system. Biodegradation by microorganisms is probably the ultimate fate process. Biodegradation generally appears to be more efficient in soil than in aquatic systems. However, it may be more important in those aquatic systems which are chronically affected by PAH contamination. Phenanthrene is stable enough in air to be transported over relatively great distances.

Health Effects

There are no epidemiological or case studies available suggesting that phenanthrene is carcinogenic in humans. This compound generally is not considered to be carcinogenic in experimental animals. However, at least two skin painting studies report development of tumors at the site of application in mice. Phenanthrene exhibits mutagenic activity in some test systems, but not in others. There are no reports of teratogenic or reproductive effects due to phenanthrene exposure.

Little information concerning acute and chronic toxic effects is available. Although specific data concerning exposure to phenanthrene are not available, workers exposed to materials containing this compound may exhibit chronic dermatitis, hyperkeratoses, and other skin disorders.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity to domestic animals and wildlife are not available. A 96-hour LC_{50} value of 600 µg/liter is reported for a saltwater polychaete worm exposed to a crude oil fraction containing phenanthrene. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 486.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

Risk	Concentration
10^{-5}	28 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

REFERENCES

NATIONAL INSTIUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1984. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. April 1984

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U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Phenanthrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H029 (Final Draft)

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2332 pages

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

Risk	Concentration
10^{-5}	28 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

REFERENCES

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1984. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. April 1984

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PHENOL

Summary

When applied to the skin of mice, phenol appears to have some tumor-promoting effects and may be a weak carcinogen. There is equivocal evidence that phenol is mutagenic. Subchronic exposure to phenol caused liver, kidney, lung, and heart damage in experimental animals. In humans, phenol has been shown to irritate the eyes, nose, and throat.

CAS Number: 108-95-2

Chemical Formula: C_6H_5OH

IUPAC Name: Phenol

Chemical and Physical Properties

Molecular Weight: 94.11

Boiling Point: 181.75°C

Melting Point: 43°C

Specific Gravity: 1.0576 at 20°C

Solubility in Water: 93,000 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, chloroform, and carbon disulfide; very soluble in ether; miscible with carbon tetrachloride and hot benzene

Log Octanol/Water Partition Coefficient: 1.46

Vapor Pressure: 0.3513 mm Hg at 25°C

Vapor Density: 3.24

pKa: 10.02

Flash Point: 85°C (closed cup)

Transport and Fate

Photooxidation may be an important degradative process, especially in aerated, clear, surface waters. Phenol may also be nonphotolytically oxidized in highly aerated waters that contain iron and copper in solution or as part of the suspended particulates. The relatively low log octanol/water partition coefficient of phenol, as well as the available experimental evidence, suggest that sorption and bioaccumulation are not important environmental fate processes. Biodegradation can be a significant fate pathway in aquatic systems and soil when significant concentrations of microorganisms are present. In addition to microorganisms, at least one species of fish is reported to be able to biotransform phenol.

The dominance of photooxidation, metal-catalyzed oxidation, or biodegradation as destructive pathways depends on the particular environmental conditions, but the degradation products are similar for all fate pathways. The first step usually involves further hydroxylation of the aromatic ring, followed by oxidation to benzoquinone and cleavage of the ring structure. There is a possibility that phenol present in surface waters can volatilize into the atmosphere. However, since this phenol would be rapidly photooxidized in the troposphere, any significant atmospheric transport is unlikely.

Health Effects

Phenol appears to have tumor-promoting activity in many strains of mice when repeatedly applied to the shaved skin after initiation with known carcinogens. Although there is equivocal evidence that phenol may be weakly carcinogenic when applied to the skin of one sensitive strain of mice, it does not appear to be carcinogenic when applied to the skin of standard strains of mice. NCI reported that phenol was not carcinogenic when administered in drinking water to rats and mice. There is equivocal evidence that phenol may have mutagenic effects, although further evaluation is needed. There are no reports of teratogenic effects caused by exposure to phenol.

Subchronic inhalation exposure to phenol is reported to cause liver, kidney, lung, and heart damage in guinea pigs. Slight liver and kidney damage was seen in rats exposed by gavage to 100 mg/kg/day for 20 days. The oral and skin LD$_{50}$s for the rat are 414 and 669 mg/kg, respectively, and the inhalation LC$_{50}$ is 316 mg/m^3. Phenol is an eye, nose, and throat irritant and can cause systemic damage to the nervous system in humans following dermal, oral, or inhalation exposure.

Toxicity to Wildlife and Domestic Animals

The acute toxicity of phenol to freshwater species is expressed over a range of 2 to 3 orders of magnitude. Acute values for fish species range from 5,020 μg/liter for juvenile rainbow trout to 67,500 μg/liter for the fathead minnow. The acute value for the rainbow trout, and a value of 5,000 μg/liter for *Daphnia magna* are the lowest acute values observed. An early life stage test on the fathead minnow resulted in a chronic value of 2,560 μg/liter, with an acute-chronic ratio of 14. Median effect concentrations for oyster and clam embryos are approximately 55,000 μg/liter. For the grass shrimp and the mountain bass, LC_{50} values of 5,800 and 11,000 μg/liter, respectively, are reported. No chronic effects are available for saltwater species. Reported bioconcentration factors of 1.2 to 2.3 for goldfish suggest that no residue problem should occur from exposure to phenol. No appropriate data concerning effects of phenol on other wildlife or domestic animals are available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, the lowest concentrations of phenol known to cause toxic effects in aquatic organisms are:

Freshwater

Acute toxicity: 10,200 μg/liter
Chronic toxicity: 2,560 μg/liter

Saltwater

Acute toxicity: 5,800 μg/liter
Chronic toxicity: No available data

Human Health

Health criterion: 3.5 mg/liter
Organoleptic criterion: 0.3 mg/liter

NIOSH Recommended Standards: 20 mg/m^3 TWA
60 mg/m^3/15 min Ceiling Level

OSHA Standard: 19 mg/m^3

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ACGIH Threshold Limit Values: 19 mg/m^3 TWA
38 mg/m^3 STEL

Department of Transportation: Poison

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NATIONAL CANCER INSTITUTE (NCI). 1980. Bioassay of Phenol for Possible Carcinogenicity. CAS No. 108-95-2. NCI Carcinogenesis Technical Report Series No. 203. Washington, D.C. USDHHS Publication No. (NTP) 80-15

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U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-066

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WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

PHENYL ETHER

Summary

Phenyl ether is somewhat persistent in the natural environment. It can cause nausea in humans. High concentrations of phenyl ether irritate the skin.

CAS Number: 101-84-8

Chemical Formula: C_6H_5-O-C_6H_5

IUPAC Name: Phenoxybenzene

Important Synonyms and Trade Names: Diphenyl ether, diphenyloxide, phenoxybenzene

Chemical and Physical Properties

Molecular Weight: 170.20

Boiling Point: 257-259°C

Melting Point: 28°C

Specific Gravity: 1.073 at 20°C

Solubility in Water: Insoluble in water

Solubility in Organics: Soluble in alcohol, benzene, ether, and glacial acetic acid

Log Octanol/Water Partition Coefficient: 4.1 (calculated)

Vapor Pressure: 0.02 mm Hg at 25°C

Vapor Density: 5.86

Flash Point: 115°C

Transport and Fate

No information on the transport and fate of phenyl ether was found in the literature reviewed, but information was available on 4-chlorophenyl phenyl ether. Based on this information and on the chemical and physical properties of phenyl ether, probable transport and fate processes can be determined.

Although phenyl ether has a low vapor pressure, its low water solubility suggests that limited volatilization is likely. Once in the atmosphere, it will be photooxidized. Phenyl ether has a high log octanol/water partition coefficient; it probably both adsorbs to soil and sediments and bioaccumulates. The results of a study on 4-chlorophenyl phenyl ether suggest that biodegradation may be important in acclimated microbial populations but not in populations in natural waters. Phenyl ether is likely to be somewhat persistent in the natural environment, with adsorption to organics acting as a storage mechanism.

Health Effects

Phenyl ether has not been reported to be carcinogenic, mutagenic, or teratogenic. A disagreeable odor and possible nausea provide sufficient warning of exposure. The undiluted material is somewhat irritating to the skin after prolonged exposure, and erythema and exfoliation are possible. However, the irritation clears promptly once exposure ceases. The oral LD_{50} for rats is 3.99 g/kg; for the guinea pig, it is approximately 2.5 g/kg.

Toxicity to Wildlife and Domestic Animals

The 96-hour LC_{50} values for phenyl ether are 9.6 mg/liter for the fathead minnow and 0.72 mg/liter for *Daphnia magna*.

Regulations and Standards

OSHA Standard (air): 7 mg/m^3 TWA

ACGIH Threshold Limit Values: 7 mg/m^3 TWA
14 mg/m^3 STEL

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

PHOSPHORIC ACID

Summary

Solutions of phosphoric acid are corrosive and are severely irritating to the skin, eyes, and mucous membranes.

CAS Number: 7664-38-2

Chemical Formula: H_3PO_4

IUPAC Name: Phosphoric acid

Important Synonyms and Trade Names: Orthophosphoric acid

Chemical and Physical Properties

Molecular Weight: 98.04

Boiling Point: Loses one-half H_2O at 213°C

Melting Point: 42.35°C

Specific Gravity: 1.834 at 18°C

Solubility in Water: Soluble in water

Solubility in Organics: Soluble in alcohol

Log Octanol/Water Partition Coefficient: -1.6 (calculated)

Vapor Pressure: 0.0285 mm Hg at 20°C

Tribasic acid: $pk_1=2$, $pk_2=7$, $pk_3=12$

Transport and Fate

No information on the transport and fate of phosphoric acid was found in the literature reviewed. Phosphoric acid probably is not very volatile. It is soluble in water, and the degree of solubility depends on the pH of the water. It may form insoluble salts and precipitate in association with such metals as iron, aluminum, and zinc. Its movement through soils is dependent on their pH and may be limited by binding to minerals.

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Health Effects

Phosphoric acid, H_3PO_4, is a tribasic acid in which the first hydrogen is strongly ionizing, the second moderately ionizing, and the third very weakly ionizing. Solutions of trihydrogen phosphate are acidic and corrosive. Phosphoric acid is stronger than acetic acid but weaker than hydrochloric, nitric, or sulfuric acid. Phosphoric acid is a severe irritant to skin, mucous membranes, and eyes; and the level of irritation is positively correlated with the degree of acidity. Phosphoric acid has no known carcinogenic, mutagenic, or teratogenic properties. The oral LD_{50} for rats is 1,530 mg/kg, while the dermal LD_{50} for rabbits is 2,740 mg/kg.

Toxicity to Wildlife and Domestic Animals

The toxicity of phosphoric acid to aquatic organisms is due to its acidic nature, and therefore the toxic concentration will depend upon the buffering capacity of the natural waters, as well as the relative resistance of each organism to low pH conditions. Phosphoric acid may be most harmful to the environment because it is an important nutrient and not because of its toxicity. Phosphate is very often the nutrient that by its absence limits the growth of algae. In the presence of sufficient phosphate, algal blooms occur which, as they die and decay, lead to eutrophication of the body of water and cause the elimination of species that need higher oxygen levels and a more pristine environment. Airborne phosphoric acid should not cause necrosis of vegetation or irritation to domestic animals at concentrations below 1 mg/m^3, but such effects are possible at higher concentrations. Phosphoric acid does not bioaccumulate in the tissues of wildlife species.

Regulations and Standards

OSHA Standard (air): 1 mg/m^3 TWA

ACGIH Threshold Limit Values: 1 mg/m^3 TWA
3 mg/m^3 STEL

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

PHOSPHORUS (WHITE)

Summary

White phosphorous is highly toxic; the oral LD$_{50}$ in the rat is 3 mg/kg. Chronic exposure to phorphorous causes bone changes, including bone necrosis. In humans, this effect occurs more often in the jaws and is termed "phossy jaw." Inhalation of phosphorous caused severe respiratory irritation and edema in rats.

CAS Number: 7723-14-0

Chemical Formula: P_4

IUPAC Name: Phosphorus

Important Synonyms and Trade Names: Yellow phosphorus, Rat-Nip

Chemical and Physical Properties

Molecular Weight: 123.88

Boiling Point: 280°C

Melting Point: 44.1°C

Specific Gravity: 1.82 at 20°C

Solubility in Water: 3 mg/liter

Solubility in Organics: Soluble in alcohol, ether, chloroform and benezene

Vapor Pressure: 0.026 mm Hg at 20°C

Vapor Density: 4.42

Flash Point: Spontaneous in air

Transport and Fate

Elemental phosphorus reacts spontaneously with air. In water it is slowly converted to phosphate with the extent of conversion dependent on the physical nature of the particular media. Phosphorus, as phosphate, is an essential nutrient for plants, and especially for aquatic plants, is often the limiting nutrient. Therefore, bioaccumulation of phosphate

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by plants is probably an important fate for phosphorus in the environment.

Health Effects

No information on the carcinogenicity or mutagenicity of phosphorus was found in the literature reviewed. Female rats exposed to as little as 11 µg/kg of phosphorus on days 1 through 22 of gestation had decreased fertility.

The most common nonfatal effects of phosphorus are bone changes, including bone necrosis. In humans, this effect occurs most often in the jaws of occupationally exposed workers and is termed "phossy jaw." Phosphorus is highly toxic, with death reported in a human who ingested a dose of 1 mg/kg. The oral LD_{50} in the rat is 3 mg/kg. Inhalation by rats of 100 mg/m^3 caused severe respiratory irritation and high mortality due to bronchopneumonia or edema.

Toxicity to Wildlife and Domestic Animals

Bluegill sunfish had 48 and 160 hour LC_{50} values of 105 µg/liter and 25 µg/liter, respectively. Adult salmon exposed to elemental phosphorus concentrations of less than 40 µg/liter show signs of extensive hemolysis. Juvenile Atlantic salmon exposed to phosphorus had a 195-hour LC_{50} of 0.8 µg/liter, and Atlantic cod had a 125-hour LC_{50} of 1.9 µg/liter. Fish bioconcentrate phosphorus to rather high levels (50 times environmental levels in muscle and 25,000 times water levels in liver) following fairly short (24 hour) exposure.

Phosphorus converted to phosphate is an environmental problem not because of its toxicity but because of its action as an essential nutrient. Low phosphate levels in the aquatic environment often beneficially limit the growth of algae and the introduction of excess phosphate causes algal blooms which reduce the oxygen in the water. This decrease in oxygen can cause the death of some fish and invertebrate species.

Phosphorus caused toxic effects in pigs exposed to a dose of 160 mg/kg and had minor behavioral effects on ducks at 3 mg/kg.

Regulations and Standards

OSHA Standard (air): 100 $\mu g/m^3$ TWA

ACGIH Threshold Limit Values: 100 $\mu g/m^3$ TWA
300 $\mu g/m^3$ STEL

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PICRIC ACID

Summary

Picric acid was mutagenic when tested using the Ames assay. In humans, exposure has been associated with nausea, abdominal pain, pruritus, and skin disorders.

CAS Number: 88-89-1

Chemical Formula: $C_6(NO_2)_3H_2OH$

IUPAC Name: 2,4,6-Trinitrophenol

Important Synonyms and Trade Names: Carbazotic acid, 2-hydroxy-1,3,5-trinitrobenzene, melinite, nitroxanthic acid, phenol trinitrate, piero nitric acid

Chemical and Physical Properties

Molecular Weight: 229.11

Boiling Point: Explodes at temperatures greater than 300°C

Melting Point: 122-123°C

Specific Gravity: 1.763 at 20°C

Solubility in Water: 14,000 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, diethyl ether, acetone, benzene, acetic acid, and pyrimidine

Log Octanol/Water Partition Coefficient: Low (approximately 2)

Vapor Pressure: 1 mm Hg at 195°C

Vapor Density: 7.91

Flash Point: 150.0°C

Transport and Fate

There is little available data on the transport and fate of picric acid. Picric acid has a relatively low vapor pressure; therefore, volatilization probably is not an important transport process.

In water, bacteria degrade picric acid by reducing the NO_2 groups to NH_2. Microbial biodegradation is probably the most important fate process for picric acid in the environment. Picric acid is transported readily by surface and groundwater (Burton et al. 1984). Its adsorption to sediments and soil probably is not significant. Picric acid has an affinity for protein, which can lead to its uptake in the tissue of aquatic organisms. For example, the American oyster (*Crassostrea virginica*) has shown biphasic uptake with retention in the tissues of approximately 50% (Burton et al. 1984).

Picric acid can be metabolized to a small extent in aquatic organisms.

Health Effects

No carcinogenic data were available in the literature reviewed. However, in a study to evaluate its mutagenic potential, picric acid yielded positive results in both the Ames assay and the Basc test using *Drosophila* (Gocke et al. 1981). No information on reproductive toxicity was available in the literature reviewed.

In humans, the ingestion or percutaneous absorption of picric acid may cause nausea, vomiting, diarrhea, abdominal pain, olicuria anuria, pruritus, and skin eruptions. Skin disease appears to be the most common toxic effect associated with exposure to picric acid. For example, an outbreak of hematuria was observed in naval personnel after the dumping of some ammunition containing the acid in their vicinity. The LD_{Lo} for picric acid administered orally ranged from 250 mg/kg in cats to 100 mg/kg in guinea pigs.

Toxicity to Wildlife and Domestic Animals

Available data for freshwater species consist of acute toxicity tests on fish, arthropods, and algae.

For fish, the LC_{Lo} was reported to be 88 mg/liter. Trout were listed as having a perturbation level of 4 g/liter after 50 minutes. Of the invertebrate species tested, *Daphnia* had an LC_{Lo} of 88 mg/liter and a 42-day study on *Crassostrea virginica* revealed that picric acid inhibits growth.

Scenedesmus showed an LC_{Lo} of 240 mg/liter. Inhibition of cell multiplication was noted at 70 mg/liter in *Microcystic aeruginosa*. There were no data available on saltwater species.

The LD_{Lo} was measured in pigeons and frogs using subcutaneous injections. It was 200 mg/kg for both, suggesting that picric acid is unlikely to be an environmental hazard to terrestrial wildlife or domestic animals. No other studies on the toxicity of picric acid were found in the literature reviewed.

Regulations and Standards

OSHA Standard (air): 100 $\mu g/m^3$ TWA (skin)

ACGIH Threshold Limit Values: 100 $\mu g/m^3$ TWA
300 $\mu g/m^3$ STEL

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POLYCHLORINATED BIPHENYLS

Summary

Polychlorinated biphenyls (PCBs) are very persistent in the natural environment and are readily bioaccumulated. In humans, exposure to PCBs has been associated with chloracne, impairment of liver function, a variety of neurobehavioral symptoms, menstrual disorders, minor birth abnormalities, and an increased incidence of cancer. Experimental animals exposed to PCBs experienced an increased incidence of cancer; reproductive problems; neurobehavioral degradation; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological function. PCBs are often contaminated, and these contaminants may be much more toxic than the PCBs themselves.

Background Information

Polychlorinated biphenyls (PCBs) are complex mixtures of chemicals composed of two connected benzene rings with 1 to 10 chlorine atoms attached. The chemical, physical, and biological properties of these materials depend to a large degree on the amount and location of the chlorine atoms on the two benzene rings of each specific PCB and on the particular mixture of individual chlorobiphenyls that comprise the mixture.

CAS Number: 1336-36-3

Chemical Formula: $C_6H_5Cl_xC_6H_5Cl_x$

IUPAC Name: Specific for each polychlorinated biphenyl

Important Synonyms and Trade Names: PCBs, chlorinated biphenyls, polychlorobiphenyls, Aroclor, Kanechlor, Clophen

Chemical and Physical Properties

Molecular Weight: 189-399*

Boiling Point: 267°C and up*

Melting Point: 54-310°C*

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*Increases with increasing chlorination.

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Specific Gravity: 1.3 to 1.5 at 20°C*

Solubility in Water: 0.003-0.6 mg/liter

Solubility in Organics: Soluble in most common organic solvents

Log Octanol/Water Partition Coefficient: 4-6*

Vapor Pressure: 10^{-3}-10^{-5} mm Hg at 20°C**

Henry's Law Constant: 10^{-3} to -10^{-5} atm m^3/mole

Transport and Fate

The transport and fate of polychlorinated biphenyls has been studied extensively, and although individual chemicals vary in the rates at which processes occur, some generalizations can be made about PCBs as a class. PCBs are relatively inert, and therefore persistent, compounds, with low vapor pressures, low water solubility, and high log octanol/water partition coefficients. Despite their low vapor pressures, they have a high activity coefficient in water, which causes a higher rate of volatilization than might normally be expected. Volatilization and persistence account for the ubiquitous nature of PCBs in the environment. Adsorption to the organic material in soil or sediments is probably the major fate of at least the more heavily chlorinated PCBs. Once bound, the PCBs may persist for years with slow desorption providing continuous, low-level exposure to the surrounding locality. Bioaccumulation of PCBs also occurs, with most of the compound stored in the adipose tissue of the body. PCBs are degraded primarily by two routes. Less heavily chlorinated PCBs (mainly the mono-, di-, and trichlorinated PCBs) can be biodegraded by some soil microorganisms. PCBs with five or more chlorines are not measurably biodegraded. These heavier PCBs can be photolyzed by ultraviolet light. This process is extremely slow, but it may be the most important degradation process for these very persistent compounds.

Assessing the toxicity of PCBs is complicated by the fact that several different mixtures have been produced and distributed commercially and by the presence of highly toxic contaminants in some commercial mixtures. Some of these contaminants can be formed by combustion of PCBs or even by high-temperature treatment in service, so that used materials may be more toxic than the commercial mixtures whose toxicity has been studied.

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*Increases with increasing chlorination.
**Decreases with increasing chlorination.

Health Effects

In humans exposed to PCBs (in the workplace or via accidental contamination of food), reported adverse effects include chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, a variety of neurobehavioral and affective symptoms, menstrual disorders, minor birth abnormalities, and probably increased incidence of cancer. Animals experimentally exposed to PCBs have shown most of the same symptoms, as well as impaired reproduction; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological functions. PCBs are carcinogenic in rats and mice and, in appropriate circumstances, enhance the effects of other carcinogens. Reproductive and neurobiological effects of PCBs have been reported in rhesus monkeys at the lowest dose level tested, 11 μg/kg body weight/day over a period of several months.

Toxicity to Wildlife and Domestic Animals

Polychlorinated biphenyls are bioaccumulated and can be biomagnified. Therefore, their toxicity increases with length of exposure and position of the exposed species on the food chain. The toxicity of the various PCB mixtures is also dependent on their composition. Because of the complexity of PCB toxicity, only general effects will be discussed here.

The 96-hour LC_{50} values for rainbow trout, bluegills, and channel catfish were around 20 mg/liter. The same species exposed for 10 to 20 days had LC_{50} values of about 0.1 mg/liter. Invertebrate species were also adversely affected, with some species having 7-day LC_{50} values as low as 1 μg/liter. In general, juvenile organisms appeared more susceptible to the effects of PCBs than either eggs or adults.

Three primary ways in which PCBs can affect terrestrial wildlife are outright mortality, adversely affecting reproduction, and changing behavior. PCB doses greater than 200 ppm in the diet or 10 mg/kg body weight (bw) caused some mortality in sensitive bird species exposed for several days. Doses around 1,500 ppm (diet) or about 100 mg/kg (bw) caused extensive mortality in these sensitive species. They generally caused some mortality in all species, with the level being dependent on the length of exposure and the particular PCB mixture. Some mammalian species are especially susceptible to PCBs. For example, mink died when fed as little as 5 ppm in the diet (equivalent to less than 1 mg/kg bw/day). PCBs caused lower egg production; deformities; decreased hatchability, growth, and survival; and some eggshell thinning in reproductive studies on chickens fed doses of 20 ppm in the diet (1 mg/kg bw). Mink fed 1 ppm in the diet (0.2 mg/kg bw) had lower reproductive success, and there are indications that an increased incidence of premature births in some marine mammals was linked to PCB exposure. Behavioral effects on wildlife include increased activity, decreased avoidance response, and decreased nesting, all of which could significantly influence survival in the wild.

No toxic effects on domestic animals other than chickens were reported in the sources reviewed, but susceptible species would probably be affected in a similar manner to laboratory animals and wildlife.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 2 μg/liter
Chronic toxicity: 0.014 μg/liter

Saltwater

Acute toxicity: 10 μg/liter
Chronic toxicity: 0.030 μg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of PCBs in water are:

Risk	Concentration
10^{-5}	0.79 ng/liter
10^{-6}	0.079 ng/liter
10^{-7}	0.0079 ng/liter

CAG Unit Risk (USEPA): 4.34 $(mg/kg/day)^{-1}$

NIOSH Recommended Standard: 1.0 $\mu g/m^3$ TWA

ACGIH Threshold Limit Value: 0.5 mg/m^3 TWA

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POLYCHLORINATED DIBENZO-p-DIOXINS

Summary

Polychlorinated dibenzo-p-dioxins (PCDDs) are often found as contaminants in chlorinated phenolic compounds. They persist in the natural environment and can be bioaccumulated. Exposure to PCDDs has been associated with numerous adverse health effects, including cancer, genotoxicity, enzyme induction; chloracne, teratogenicity, reproductive toxicity, immunotoxicity, porphyria cutanea tardo, and neurobehavioral changes.

Chemical Formula: $C_{12}H_4Cl_xO_2$

IUPAC Name: Polychlorodibenzo-1,4-dioxins

Important Synonyms and Trade Names: Dioxins, PCDDs

Chemical and Physical Properties

Boiling Point: 500°C (begins to decompose)

Melting Point: Around 300°C

Solubility in Water: Insoluble

Solubility in Organics: Soluble in fats, oils, and other relatively nonpolar solvents

Log Octanol/Water Partition Coefficient: Approximately 5

Vapor Pressure: 10^{-6} mm Hg at 25°C

Transport and Fate

Polychlorinated dibenzo-p-dioxins (PCDDs) have a very low vapor pressure and therefore are unlikely to volatize into the atmosphere. However, there are studies that indicate volatization may occur. Experiments have shown PCDDs to be highly sorbed to sediments, soils, and bioata so they may be transported through the air in soil dust. Because PCDDs are tightly bound to soils, it is probable that any surface water contamination found in polluted areas is from soil erosion rather than from leaching. A calculated sediment/water equilibrium partition coefficient using six sets of data for PCDDs varied from 1.1×10^{-4} to 2.1×10^{-4}. This indicates that most PCDDs in water will be sorbed to particulates.

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PCDDs do not readily undergo photodegradation unless solvents are present which will act as hydrogen donors during reductive dechlorination. Certain microorganisms have been found that will degrade PCDDs. The half-life of PCDDs in soil has been found to vary from 130 days to several years. Thus, PCDDs are persistent in the environment.

Health Effects

Studies of the health effects of the PCDDs have generally concentrated on 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) because it is the most toxic of the PCDDs. Studies on the other PCDDs indicate that they cause the same effects, but at different quantitative doses than TCDD.

The structure-activity relationships among the PCDDs are reasonable well-defined. Isomers with 2 or fewer chlorine atoms in the 2, 3, 7, and 8 positions have low biological activity. Isomers with 3 or 4 of these positions substituted have quantitativel and qualitatively similar to biological activity TCDD. The 1,2,3,7, and 8 positions are all only slightly less active than TCDD. Additional substitutions in the 1, 4, 6, and 9 positions considerably reduce biological activity.

A variety of health effects have been associated or attributed to exposure to very low concentrations of PCDDs, especially TCDD in both experimental animals and humans. These effects include cancer, genotoxicity, enzyme induction, teratogenicity and reproductive toxicity, immunotoxicity, chloracne, porphyria cutanea tarda, and neurobehavioral toxicity. TCDD has been shown to induce cancer in mice and rats following dermal or oral administration. Animal studies suggest that immunotoxicity is probably the most potent effect of TCDD. Both immunotoxicity and the enzyme inducing effects of PCDDs are probably mediated through a cytosolic receptor that high affinity for PCDDs. Chloracne is the only clear effect that PCDD intoxication has produced in humans.

Toxicity to Wildlife and Domestic Animals

Freshwater aquatic species exposed to low concentrations of TCDD (in the parts per trillion range) for 4 days displayed toxic signs and died from 40 to 140 days later. Acute toxic effects were not noted in many of the aquatic species at the level of TCDD water solubility, 0.2 μg/liter. Horses exposed to TCDD in contaminated waste oil used to control dust in corrals, became sick and died.

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POLYCYCLIC AROMATIC HYDROCARBONS

Summary

Polycyclic aromatic hydrocarbons (PAHs) are rather persistent in the environment. Some PAHs are carcinogenic, causing tumors both at the site of application and systemically. The carcinogenic PAHs are generally active in mutagenic assays. They also cause skin disorders and immunosuppression. Adverse effects on the liver and kidney have been associated with exposure to PAHs in general.

Important Synonyms and Trade Names: Polynuclear aromatic hydrocarbons, PAH, PNA

Chemical and Physical Properties

The polycyclic aromatic hydrocarbons are a class of compounds consisting of substituted and unsubstituted polycyclic aromatic rings formed by the incomplete combustion of organic materials. Their chemical, physical, and biological properties vary with their size and shape.

Molecular Weight: 116-278

Melting Point: 80°C-270°C*

Specific Gravity: 1.1-1.3 at 20°C*

Solubility in Water: 0.0003-34 mg/liter**

Solubility in Organics: Soluble in most common organic solvents

Log Octanol/Water Partition Coefficient: 3.4-7.6*

Vapor Pressure: 10^{-10} to 10^{-2} mm Hg at 20°C**

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*Generally increases with increasing molecular weight.

**Generally decreases with increasing molecular weight.

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Transport and Fate

Very little information on specific polycyclic aromatic hydrocarbons (PAHs) is available. The environmental fate and transport of these compounds are largely inferred from data on benzo(a)pyrene and mixtures of PAHs. The relatively high log octanol/water partition coefficients of PAHs indicate that they should be strongly adsorbed onto suspended particulate matter, especially particulates high in organic content. The available information suggests that these compounds can accumulate in the sediment and biota portions of the aquatic environment and that adsorption is probably the dominant aquatic transport process. Atmospheric transport of PAHs is also possible. This generally occurs by adsorbtion onto airborne particulate matter, but some of the PAHs with relatively low molecular weights are volatile. Regardless of the method of atmospheric transport, PAHs are returned to aquatic and terrestrial systems by atmospheric fallout or precipitation. They can also reach ground or surface waters by leaching from polluted soils.

PAHs are relatively insoluble in water, but the dissolved portion may undergo rapid, direct photolysis. Singlet oxygen is the oxidant, and quinones are the products in these reactions. Oxidation by chlorine and ozone may be an important fate process when these oxidants are available in sufficient quantities.

Although polycyclic aromatic hydrocarbons are rapidly bioaccumulated, they are also quickly metabolized and eliminated from most organisms (shellfish are a known exception). Bioaccumulation, especially in vertebrate organisms, is usually short term, so it is not considered an important fate process in multicellular organisms. Biodegration and biotransformation are probably the ultimate fate processes for PAHs. The available data suggest that the PAHs with high molecular weights are degraded slowly by microbes and readily metabolized by multicellular organisms. Microbes appear to degrade PAHs much more completely than mammals. Biodegradation probably occurs more slowly in aquatic systems than in soil, and it may be much more important in systems that are chronically affected by PAH contamination.

Health Effects

The potential for PAHs to induce malignant transformation dominates the consideration of health hazards resulting from exposure, because there often are no overt signs of toxicity until the dose is high enough to produce a high tumor incidence. The attached table contains IARC's classification of some PAHs according to their carcinogenicity.

No case reports or epidemiological studies concerning the significance of human exposure to individual PAHs are available.

However, coal tar and other materials known to be carcinogenic to humans contain PAHs.

PAHs administered by various routes have been found to be carcinogenic in several animal species and to have both local and systemic carcinogenic effects. On oral administration, carcinogenic PAHs produce tumors of the forestomach in mice. Lung tumors are produced in hamsters after intratracheal administration and in mice after intravenous administration. In skin painting experiments with mice, carcinogenic PAHs produced skin carcinomas. Other observed effects include induction of local sarcomas and an increased incidence of lung adenomas in mice following single, subcutaneous injections. Studies in other species, while indicating that PAHs have universal carcinogenic effects, are less complete. Carcinogenic PAHs are reported to be mutagenic in a variety of test systems. The limited available information suggests that PAHs are not very potent teratogens or reproductive toxins.

There is very little information regarding nonmalignant changes caused by exposure to PAHs. Application of carcinogenic PAHs to mouse skin is reported to cause destruction of sebaceous glands, hyperplasia, hyperkeratosis, and ulceration. Many carcinogenic PAHs also have immunosuppressive effects. Subcutaneous injections of some PAHs for several weeks reportedly caused hemolymphatic changes in the lymph nodes in rats. Workers exposed to PAH-containing materials have exhibited chronic dermatitis, hyperkeratoses, and other skin disorders.

Toxicity to Wildlife and Domestic Animals

There is very little information on the environmental toxicity of PAHs; they probably are not very toxic to aquatic organisms.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

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Risk	Concentration
10^{-5}	28.0 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

CAG Unit Risk (USEPA): Benzo(a)pyrene: 11.5 $(mg/kg/day)^{-1}$

CARCINOGENICITY OF PAHs

Chemicals for which there is sufficient evidence that they are carcinogenic in animals:

Benzo(a)anthracene
Benzo(b)fluoranthene
Benzo(j)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Dibenzo(a,h)acridine
Dibenzo(a,j)acridine
Dibenzo(a,h)anthracene
7H-Dibenzo(c,g)carbazole
Dibenzo(a,e)pyrene
Dibenzo(a,h)pyrene
Dibenzo(a,i)pyrene
Dibenzo(a,l)pyrene
Indeno(1,2,3-c,d)pyrene
5-Methylchrysene

Chemicals for which there is limited evidence that they are carcinogenic in animals:

Anthranthrene
Benzo(c)acridine
Carbazole
Chrysene
Cyclopenta(c,d)pyrene
Dibenzo(a,c)anthracene
Dibenzo(a,j)anthracene
Dibenzo(a,e)fluoranthene
2-, 3-, 4-, and 6-Methylchrysene
2- and 3-Methylfluoranthene

Chemicals for which the evidence is inadequate to assess their carcinogenicity:

Benzo(a)acridine
Benzo(g,h,i)fluoranthene
Benzo(a)fluorene
Benzo(b)fluorene
Benzo(c)fluorene
Benzo(g,h,i)perylene
Benzo(c)phenanthrene
Benzo(e)pyrene
Coronene
1,4-Dimethylphenanthrene
Fluorene
1-Methylchrysene
1-Methylphenanthrene
Perylene
Phenanthrene
Triphenylene

Chemicals for which the available data provide no evidence that they are carcinogenic:

Anthracene
Fluoranthene
Pyrene

SOURCE: IARC 1983

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SELENIUM

Summary

Although selenium is an essential element, exposure to amounts just slightly above the required levels can produce toxic effects. Signs of chronic exposure in humans include dermatitis, neurobehavioral effects, gastrointestinal disturbances, dental caries and discoloration, and partial loss of hair and nails. Toxic effects observed in animals include degeneration of the liver, kidneys, and myocardia; hemorrhages in the digestive tract; and brain damage. Inhalation of selenium irritates the eyes, nose, and throat.

Background Information

Selenium is stable in four valence states: -2, 0, +4, and +6. Elemental selenium can be considered inert in the aquatic environment, and deposition of this form appears to be a major sink for selenium in natural systems.

CAS Number: 7782-49-2

Chemical Formula: Se

IUPAC Name: Selenium

Chemical and Physical Properties

Atomic Weight: 78.96

Boiling Point: 684.9°C

Melting Point: 217°C

Specific Gravity: 4.26 to 4.81

Solubility in Water: Insoluble

Solubility in Organics: Crystals slightly soluble in carbon disulfide, soluble in ether; amorphous forms soluble in carbon disulfide, methylene iodide, benzene, and quinoline

Transport and Fate

In aerobic waters and at high pH, selenium is present in the selenite (+4) or selenate (+6) oxidation state. These chemical species are very soluble, and it is probable that most of the selenium released into the aquatic environment is transported in these forms to the oceans. Under reducing conditions and at low pH, elemental selenium or metal selenides can be formed. Similar chemical speciation patterns affect the transport of selenium in soil. In poorly aerated, acidic soils, insoluble forms predominate. In well-aerated, alkaline soils, soluble forms of selenium subject to leaching and compounds readily taken up by plants tend to be formed.

Selenium is strongly adsorbed to hydrous metal oxides, while clays and organic materials have a lesser affinity. Sorption by bed sediments or suspended solids, and precipitation with hydrous iron oxides are probably the major control on mobility of selenium in aerobic waters. However, most selenium in aquatic systems is probably transported as the dissolved species. Experimental studies indicate that selenium is quite mobile in clays, especially under alkaline conditions.

Selenium is bioaccumulated by aquatic and terrestrial organisms. Although dietary intake is thought to be the most important source of selenium in many organisms, little biomagnification appears to take place. Conversion of selenium to inert and insoluble forms may occur in terrestrial and aquatic organisms. However, selenium can be methylated by a variety of organisms, including benthic microflora. In a reducing environment, hydrogen selenide (H_2Se) may be formed. Both the methylated forms and H_2Se are volatile, and can be released to the atmosphere. Consequently, remobilization of selenium from aquatic and terrestrial systems, through biotransformation to volatile forms and subsequent atmospheric transport, can result in significant recycling.

Health Effects

There is no evidence that selenium is carcinogenic in humans. Selenium has been tested by the oral route in experimental animals, but the available data are insufficient to allow unequivocal evaluation of its carcinogenic potential. However, recent reports suggest that selenium is not carcinogenic. Several studies have shown that selenium may actually reduce the incidence of tumors under certain conditions. Mutagenicity, teratogenicity, and reproductive effects have not been adequately tested.

Selenium is an essential element in animals and probably in humans. However, exposure to amounts only slightly above the required levels can produce acute and chronic toxic effects. Acute toxicities of selenium compounds vary greatly, while the chronic effects of most forms are similar. Exposure may be by oral, inhalation, or dermal routes, and effects in humans and experimental animals are similar. Acute effects include degeneration of liver, kidneys, and myocardia, hemorrhages in the digestive tract, and brain damage. Eye, nose, and throat irritation may also occur with inhalation exposure. The acute oral LD_{50} value of sodium selenite in rats was approximately 10 mg/kg. Chronic toxicity in humans appears to occur only in areas where foods containing excessive concentrations of selenium are ingested. Signs of chronic intoxication include depression, nervousness, dermatitis, gastrointestinal disturbances, dental caries and discoloration, lassitude, and partial loss of hair and nails.

Toxicity to Wildlife and Domestic Animals

Some food and forage crops growing on certain seleniferous soils can accumulate selenium to concentrations as high as 1,000 ppm. Chronic selenium toxicity can occur in grazing animals that consume plants containing 3 to 25 ppm over a long period of time. Symptoms of chronic poisoning ("alkali" disease) include lack of vitality, loss of hair, sterility, hoof deformity, lameness, anemia, and fatty necrosis of the liver. Acute toxic effects including impairment of vision, weakness of limbs, and respiratory failure may occur in livestock consuming 100 to 1,000 ppm of selenium. There are reports that consumption of plants containing 400 to 800 ppm has been lethal to sheep, hogs, and calves. There are no reports of increased cancer rates among livestock in seleniferous areas.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Selenite)

Freshwater

Acute toxicity: 260 µg/liter
Chronic toxicity: 35 µg/liter

Saltwater

Acute toxicity: 410 µg/liter
Chronic toxicity: 54 µg/liter

No criteria for the protection of aquatic life were established for selenate.

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Human Health

Criterion: 10 μg/liter

Primary Drinking Water Standard: 0.01 mg/liter

NIOSH Recommended Standard: 0.2 mg/m^3 TWA (Se compounds, as Se)

OSHA Standard: 0.2 mg/m^3 TWA (Se compounds, as Se)

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SILVER

Summary

Exposure to high levels of silver can cause argyria (an impregnation of the tissues) and lesions of the liver, kidney, bone marrow, and lungs in humans. Liver and kidney damage, central nervous system effects, and pulmonary edema and congestion have been reported in experimental animals exposed to various silver compounds.

CAS Number: 7440-22-4

Chemical Formula: Ag

IUPAC Name: Silver

Chemical and Physical Properties

Atomic Weight: 107.868

Boiling Point: 2212°C

Melting Point: 961.93°C

Specific Gravity: 10.5 at 20°C

Solubility in Water: Insoluble (some compounds are soluble)

Solubility in Organics: Soluble in alkali cyanide solutions

Transport and Fate

Silver can exist in several chemical forms in aqueous systems. Metallic silver, which has very low solubility, is stable over much of the Eh-pH range for water. Concentrations of hydrated silver cations, usually present as the univalent species, may be controlled by reaction with chloride, bromide, and iodide ions to give insoluble silver halides. Precipitation of AgCl may exert a major control on solubility of silver where chloride concentrations are relatively high. Under the reducing conditions often found in bed sediments, formation of insoluble silver sulfides and metallic silver may also control levels of soluble silver species. Silver is strongly sorbed by manganese dioxide, ferric hydroxide, and clay minerals. Sorption is probably the dominant process leading to removal of dissolved

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silver from the water column. In general, concentrations of silver are higher in the bed sediments than in overlying waters. For example, these concentrations were reported to differ by a factor of 1,000 in an alpine lake.

Bioaccumulation of silver by aquatic plants, invertebrates, and vertebrates occurs readily and appears to depend primarily on sorption/desorption from sediments. However, the amount of silver partitioned to the biota appears to be minor in comparison with the amount partitioned to the sediments. Little food-chain magnification seems to occur. Photolysis, volatilization, atmospheric transport, and biotransformation do not appear to be important fate or transport processes for silver.

Health Effects

Only equivocal evidence exists to suggest that silver has carcinogenic activity in experimental animals. Silver implants and injected colloidal suspensions are reported to produce tumors or hyperplasia at the site of application in several studies. However, it is suggested that the effects are due to the physical form of the metal or to its action as an exogenous irritant. There are no studies to suggest that silver is carcinogenic in humans. Silver does not appear to have significant mutagenic or teratogenic activity in humans or experimental animals.

Silver can be absorbed in humans by inhalation or ingestion. The most common and most noticeable effects of excessive absorption are a local or generalized impregnation of the tissues referred to as argyria. In cases of argyria, accumulation of silver can result in a blue-gray pigmentation of the skin, hair, internal organs, and conjunctiva of the eye. Large oral doses of silver compounds may produce serious effects in humans. For example, silver nitrate can cause violent abdominal pain, vomiting, and convulsions, and ingestion of 10 grams is reported to usually be fatal. Lesions of the liver, kidney, bone marrow, and lungs have also been attributed to industrial or medicinal exposure.

Intravenous administration of silver nitrate is reported to produce pulmonary edema and congestion in experimental animals. Liver and kidney damage, central nervous system effects, and death have also been reported in experimental animals exposed to various silver compounds. The intraperitoneal LD_{50} (30 days) for Ag^+ as the nitrate in male Swiss albino mice is 13.9 mg/kg. Rats exposed to silver in their drinking water for 11 months showed no toxic effects at concentrations less than 0.4 mg/liter. Hemorrhaging occurred in the kidneys at 0.4 mg/liter. Conditioned reflex activity and immunological resistance were lowered, and brain nucleic acid content was increased at 0.5 mg/liter.

Numerous physiological changes, including growth depression, and pathomorphological changes in the liver, kidney, stomach, and small intestine were evident in rats exposed to 20 mg/liter for 5 months.

Toxicity to Wildlife and Domestic Animals

Acute toxicity values for freshwater invertebrates range from 0.25 μg/liter for *Daphnia magna* to 4,500 μg/liter for the scud *Gammarus pseudolimnaeus*. Acute values for fish range from 3.9 μg/liter for the fathead minnow in soft water to 280 μg/liter for rainbow trout in hard water. In fresh water, the acute toxicity of silver appears to decrease as hardness increases. Soluble compounds, such as silver nitrate, are generally much more toxic than insoluble compounds. Chronic values ranging from 2.6 to 29 μg/liter are reported for *Daphnia magna*. Two early life stage studies with rainbow trout report chronic values of 0.12 μg/liter. Acute-chronic ratios for *Daphnia magna* and rainbow trout are 2.0 and 54, respectively. Fresh water aquatic plants appear to be more resistant to silver than the more sensitive animals.

Acute values for saltwater organisms range from 4.7 μg/liter for the summer flounder to 1,400 μg/liter for the sheepshead minnow. A chronic value of 18 μg/liter, and an acute-chronic ratio of 14 is reported for the mysid shrimp.

Reduced cell numbers are observed in the saltwater alga *Skeletonema costatum* after exposure to 130 μg/liter of silver.

Excess silver can induce selenium, vitamin E, and copper deficiency symptoms in animals fed adequate diets, and can aggravate deficiency symptoms in animals whose diets lack one or more of these nutrients. These effects are reported in dogs, sheep, pigs, chicks, turkey poults, and ducklings.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(1.72\ [\ln(\text{hardness})] - 6.52)}$ μg/liter
Chronic toxicity: No criteria have been established

Saltwater

Acute toxicity: 2.3 μg/liter
Chronic toxicity: No criteria have been established

Human Health

Criterion: 50 μg/liter

Primary Drinking Water Standard: 50 μg/liter

OSHA Standard: 10 $\mu g/m^3$ TWA

ACGIH Threshold Limit Values: 0.1 mg/m^3 (metal)
0.01 mg/m^3 (soluble compounds)

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SODIUM

Summary

High doses of certain sodium compounds are reported to have teratogenic and reproductive effects in animals. Several studies suggest that brain damage and sudden unexpected death in human infants may be induced by high sodium levels. Exposure to high levels of sodium has also been associated with age-related increases in high blood pressure in genetically susceptible individuals.

Background Information

Sodium is the sixth most abundant element on earth. It is very reactive and is never found free in nature. It reacts violently with water, decomposing it with the evolution of H_2 and the formation of NaOH. Sodium normally does not ignite in air at temperatures below 115°C, but it may ignite spontaneousl on water. Because of its reactivity, sodium must be handled with great care, and contact between it and water and other substances with which it reacts should be avoided.

CAS Number: 7440-23-5

Chemical Formula: Na

IUPAC Name: Sodium

Chemical and Physical Properties

Atomic Weight: 22.9898

Boiling Point: 882.9°C

Melting Point: 97.81°C

Specific Gravity: 0.97

Solubility in Water: Metal decomposes explosively in water; many sodium compounds are soluble

Transport and Fate

Many sodium compounds are soluble in water, and the sodium ion is readily transported in surface water, soil, and groundwater. The extent of sodium transport in soil is dependent, to some extent, on the cation exchange capacity of the soil. Atmospheric transport of sodium occurs readily. Evaporation of ocean spray particles and their subsequent incorporation into precipitation is an important sodium cycling process. Sodium is ubiquitous in nature and is an important component of all ecosystems.

Health Effects

There is no evidence to suggest that sodium has carcinogenic or mutagenic effects in humans or experimental animals. Sodium chloride is reported to produce teratogenic and reproductive effects in experimental animals exposed to high doses by various routes. For example, mice exposed subcutaneously to over 2,000 mg/kg of sodium chloride on day 10 or 11 of gestation had an increased incidence of dead or resorbed young. Live young in this study had decreased body weights and an increased incidence of appendicular malformations, such as clubfoot and deviation of the digits (Nishimuri and Miyamoto 1969).

In humans, adverse effects of sodium occur as a result of ingestion of excess amounts of this element. Acute effects appear to occur only in neonates and young infants. Several studies suggest that permanent brain damage and sudden, unexpected deaths of infants between the ages of 2 weeks and 2 years may be due to hypernatremia. Sodium produces toxic effects and can cause death in experimental animals exposed to high concentrations. For example, the oral LD_{50} value for NaCl in rats is 3,000 mg/kg.

Clinical and epidemiological studies suggest that ingestion of excess sodium may contribute to the development of age-related increases in blood pressure and hypertension in genetically susceptible persons. Studies with experimental animals support the contention that excess sodium ingestion is related to the development of hypertension. It is estimated that at least 40 percent of the population would benefit if consumption of sidum were limited to 2,000 mg/day or less. The sodium present in drinking water contributes to the total daily intake of this element. One survey, which sampled the water supplies used by about half of the U.S. population, reported sodium ion concentrations ranging from 0.4 to 1,900 mg/liter.

Toxicity to Wildlife and Domestic Animals

Although few studies documenting effects are available, high concentrations of sodium chloride probably have detrimental effects on aquatic organisms and terrestrial plants. In lakes, increased salinity will cause stratification and thereby delay the spring turnover that oxygenates the lower levels of the lake. In addition, salinity changes due to high sodium chloride concentrations may adversely affect aquatic systems by changing the osmotic pressure and by increasing the mobility of some heavy metals such as mercury. In terrestrial systems, high sodium chloride concentrations caused by road deicing have proved fatal to roadside vegetation, and the increased soil salinity associated with irrigation has rendered cropland unusable.

Regulations and Standards

Department of Transportation: Flammable solid; dangerous when wet

REFERENCES

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SODIUM CHLORATE

Summary

Sodium chlorate yielded positive results in two mutagenicity assays. It is a strong oxidizing agent and consequently is a fairly potent irritant.

CAS Number: 7775-09-9

Chemical Formula: $NaClO_3$

IUPAC Name: Sodium chlorate

Important Synonyms and Trade Names: Asex, Atlacide, chlorate of soda, chlorate salt, Klorex Kusatol, soda chlorate

Chemical and Physical Properties

Molecular Weight: 106.44

Boiling Point: Decomposes at 300°C

Melting Point: 248°C to 261°C

Specific Gravity: 2.5 at 20°C

Solubility in Water: Approximately 1,000 mg/liter

Solubility in Organics: Soluble in alcohol and glycerol

Transport and Fate

No information on the transport and fate of sodium chlorate was found in the sources reviewed. Sodium chlorate is a strong oxidizing agent and probably reacts quite rapidly in the environment. It is not likely to be persistent in nature.

Health Effects

No information on the carcinogenicity of sodium chlorate was found in the sources reviewed. Gocke et al. (1981) reported that the results of two mutagenicity assays--the Ames assay and the BASC test on *Drosophila*--were positive for sodium chlorate. No information on the chronic toxicity of sodium chlorate was available in the sources reviewed. Sodium chlorate is a strong oxidizing agent and therefore is a fairly strong

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irritant. The oral LD_{50} in rats is 1,200 mg/kg, but doses of around 200 mg/kg were fatal to human children.

Toxicity to Wildlife and Domestic Animals

Sodium chlorate was reported to be toxic to fish, but no dose levels were provided. This compound is a nonselective herbicide. No other information on the toxicity of sodium chlorate to wildlife and domestic animals was found in the sources reviewed.

Regulations and Standards

No regulations or standards for sodium chlorate have been established.

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DOULL, J., KLAASSEN, C.D., and AMDUR, M.O., eds. 1980. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. 778 pages

GOCKE, E., KING., M.T., ECKHARDT, K., and WILD, D. 1981. Mutagenicity of cosmetic ingredients licensed by the European Communities. Mutat. Res. 90:91-109

HERBICIDE HANDBOOK OF THE WEED SCIENCE SOCIETY OF AMERICA. 1979. 4th ed. WSSA Herbicide Handbook Committee, Champaign, Illinois. Pp. 416-418

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STODDARD SOLVENT

Summary

Stoddard solvent is an eye, nose, and throat irritant in humans. Acute exposure to high vapor concentrations can cause headaches and produce narcotic effects. Chronic exposure to high airborne concentrations of Stoddard solvent may produce kidney damage.

Background Information

Stoddard solvent is a mixture of 9 to 11 carbon straight and branched paraffins, cycloparaffins, and aromatic hydrocarbons. Properties of a specific sample will depend on the composition of the particular mixture.

CAS Number: 8052-41-3

Chemical Formula: Predominant molecular species, C9-C11; 30-50% straight and branched chain paraffins, 30-40% cycloparaffins, and 10-20% aromatic hydrocarbons

Chemical and Physical Properties

Molecular Weight: 135-145 (average)

Boiling Point: 160-210°C

Specific Gravity: 0.75-0.80

Solubility in Water: Insoluble

Solubility in Organics: Miscible with most organic solvents

Vapor Pressure: 4.0-4.5 mm Hg at 25°C

Vapor Density: 5

Flash Point: 37.8-60°C

Transport and Fate

Practically no information concerning the environmental transport and fate of Stoddard solvent is available. This solvent contains a mixture of organic compounds, and each of these components may behave somewhat differently in the environment.

In general, the low water solubility and moderate vapor pressure of Stoddard solvent suggest that volatilization may be a significant transport process. Although direct oxidation in water is unlikely, photooxidation of compounds reaching the atmosphere may occur. The solubility of Stoddard solvent in organics and its low water solubility suggest that sorption to suspended particles and bed sediments containing organic components may be a significant transport process in aquatic systems. Sorption to organic materials may limit the movement of Stoddard solvent in soil. Data concerning other related hydrocarbons suggest that biodegradation by a variety of microorganisms may be an important environmental fate for Stoddard solvent, but that bioaccumulation would not be an important long-term process.

Photolysis and hydrolysis are not likely to be significant environmental fates.

Health Effects

There are no reports of carcinogenicity, mutagenicity, teratogenicity, or reproductive effects associated with exposure to Stoddard solvent. However, benzene, a potential human leukemogenic agent, may be a contaminant of some samples of refined petroleum solvents. Stoddard solvent generally contains 0.1% benzene or less, and it is thought that ordinary use of solvents containing less than 5% benzene would not produce a benzene exposure hazard.

Stoddard solvent is an eye, nose, and throat irritant in humans and has a defatting and irritating action on the skin. At relatively high vapor concentrations it can cause headaches and produce narcotic effects. Aspiration of the liquid can produce diffused chemical irritation of the lungs, resulting in edema; and a few milliliters may be fatal in these incidents. Inhalation exposure of laboratory animals can result in irritation and narcotic effects. Chronic exposure to relatively high concentrations (greater than 1,000 mg/m^3) may produce kidney damage, although these results are equivocal.

Toxicity to Wildlife and Domestic Animals

Adequate information to characterize the toxicity of Stoddard solvent to wildlife and domestic animals is not available.

Regulations and Standards

NIOSH Recommended Standard:	350 mg/m^3 TWA 1,800 mg/m^3/15 min Ceiling Level

OSHA Standard: 2,950 mg/m^3 TWA

ACGIH Threshold Limit Values: 525 mg/m^3 TWA
1,050 mg/m^3 STEL

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1,2,4,5-TETRACHLOROBENZENE

Summary

1,2,4,5-Tetrachlorobenzene caused slight liver toxicity and disrupted conditioned reflexes in studies on experimental animals. Tetrachlorobenzenes in general appear to induce the activity of microsomal enzymes.

CAS Number: 95-94-3

Chemical Formula: $C_6H_2Cl_4$

IUPAC Name: 1,2,4,5-Tetrachlorobenzene

Important Synonyms and Trade Names: Benzene tetrachloride

Chemical and Physical Properties

Molecular Weight: 215.9

Boiling Point: 245°C

Melting Point: 139.5°C

Specific Gravity: 1.734

Solubility in Water: Insoluble (probably less than 30 mg/liter)

Solubility in Organics: Soluble in ether, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 4.93

Vapor Pressure: Less than 0.1 mm Hg at 25°C

Vapor Density: 7.4

Flash Point: 155°C

Transport and Fate

Much of the information concerning transport and fate is inferred from data for chlorinated benzenes in general because specific information for 1,2,4,5-tetrachlorobenzene is lacking. Although 1,2,4,5-tetrachlorobenzene has a relatively high boiling point and low vapor pressure, data for other chlorinated benzenes suggest that volatilization may be an important transport process under some conditions. The low solubility and high activity coefficients of these compounds in aqueous solution may account for their unexpectedly high volatility. Oxidation of 1,2,4,5-tetrachlorobenzene in aquatic systems is unlikely. However, some photodegradation by hydroxyl radicals in the atmosphere may occur. Photolysis and hydrolysis are unlikely environmental fates.

The high log octanol/water partition coefficient for 1,2,4,5-tetrachlorobenzene suggests that adsorption by organic soil particles and by suspended and sedimentary organic materials in aquatic environments is probably an important environmental process. It is also likely that significant accumulation in the tissues of living organisms occurs. Although biodegradation may occur, it probably would proceed very slowly.

Health Effects

There are no reports of carcinogenic, teratogenic, or mutagenic activity by 1,2,4,5-tetrachlorobenzene in humans or experimental organisms.

Rats receiving as little as 0.005 mg/kg/day orally for up to 8 months are reported to show a disruption of conditioned reflexes and increased liver weights. Rabbits treated with 0.05 mg/kg/day show liver glycogen-forming disorders after about 6 months. In beagles, administration of 5 mg/kg/day in the diet is reported to cause a slight elevation of serum alkaline phosphatase activity and bilirubin levels after 24 months. In this study, the serum chemistry values returned to normal within 3 months after cessation of exposure, and gross and histopathological changes conducted 20 months after cessation of exposure revealed no treatment related changes. The oral LD_{50} values for rats and mice are 1,500 and 1,035 mg/kg, respectively.

Tetrachlorobenzenes appear to induce microsomal enzymes and, therefore, could increase the metabolism of compounds acted on by the cytochrome P-450 system. This could either increase or decrease the toxicity of the compound depending on whether the metabolite was more or less active than the parent material.

Toxicity to Domestic Animals and Wildlife

An acute value for 1,2,4,5-tetrachlorobenzene of 1,550 µg/liter is reported for the bluegill, a freshwater fish. No freshwater chronic values are available. The 96-hour EC_{50}s for chlorophyll a and cell numbers are 52,900 and 46,800 µg/liter, respectively for the freshwater alga Selenastrum capricornutum. Among saltwater organisms, acute values for mysid shrimp and the sheepshead minnow are 1,480 and 840 μg/liter, respectively. A chronic value of 129 μg/liter and an acute-chronic ratio of 6.5 are reported for the sheepshead minnow. The 96-hour EC$_{50}$s for chlorophyll a and cell numbers are 7,100 and 7,320 μg/liter, respectively, for the saltwater alga Skeletonema costatum.

The weighted average bioconcentration factor for 1,2,4,5-tetrachlorobenzene and the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is estimated to be 1,125.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 38 μg/liter

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2,3,7,8-TETRACHLORODIBENZO-p-DIOXIN

Summary

Tetrachlorodibenzo-p-dioxin (TCDD) is often found as a contaminant in chlorinated phenolic compounds. It persists in the natural environment and can be bioaccumulated. Exposure to TCDD has been associated with numerous adverse health effects, including cancer, genotoxicity, enzyme induction, chloracne, teratogenicity, reproductive toxicity, immunotoxicity, porphyria cutanea tarda, and neurobehavioral changes.

CAS Number: 1746-01-6

Chemical Formula: $C_{12}H_4Cl_4O_2$

IUPAC Name: 2,3,7,8-Tetrachlorodibenzo-1,4-dioxin

Important Synonyms and Trade Names: Dioxin, TCDD, 2,3,7,8-TCDD, 2,3,7,8-tetrachlorodibenzo(b,e)-(1,4)dioxin, tetradioxin

Chemical and Physical Properties

Molecular Weight: 321.9

Boiling Point: 500°C (begins to decompose)
800°C (virtually complete degradation)

Melting Point: 302-305°C

Solubility in Water: 0.2 µg/liter at 20°C

Solubility in Organics: Soluble in fats, oils, and other relatively nonpolar solvents

Log Octanol/Water Partition Coefficient: 5.16 (measured)

Vapor Pressure: 10^{-6} mm Hg at 25°C

Henry's Law Constant: 2.1×10^{-3} atm m^3 mol^{-1}

Transport and Fate

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) has a very low vapor pressure and therefore is unlikely to volatize into the atmosphere. However, there are studies that indicate volatization may occur. Experiments have shown TCDD to be highly sorbed to sediments, soils, and biota so it may be transported through the air in soil dust. Because TCDD is tightly bound to soils, it is probable that any surface water contamination found in polluted areas is from soil erosion rather than from leaching. A calculated sediment/water equilibrium partition coefficient using 6 sets of data for TCDD varied from 1.1 x 10^{-4} to 2.1 x 10^{-4}. This indicates that most TCDD in water will be sorbed to particulates.

TCDD does not readily undergo photodegradation unless solvents are present that will act as hydrogen donors during reductive dechlorination. Certain microorganisms have been found that will degrade TCDD. The half-life of TCDD in soil has been found to vary from 130 days to well over a year. Thus, TCDD is persistent in the environment.

Health Effects

A variety of health effects have been associated or attributed to exposure to very low concentrations of TCDD in both experimental animals and humans. These effects include cancer, genotoxicity, enzyme induction, teratogenicity and reproductive toxicity, immunotoxicity, chloracne, porphyria cutanea tarda, and neurobehavioral toxicity. TCDD has been shown to induce cancer in mice and rats following dermal or oral administration. Epidemiological studies on exposed populations provide suggestive, but not conclusive, evidence that TCDD is a carcinogen in humans. There is strong evidence that TCDD is teratogenic to certain animal species, however, the evidence on humans is weak. Animal studies suggest that immunotoxicity is probably the most potent effect of TCDD. Both immunotoxicity and the enzyme inducing effect of TCDD are probably mediated through a cytosolic receptor with high affinity for TCDD. Chloracne is the only clear effect that TCDD intoxication has produced in humans.

Toxicity to Wildlife and Domestic Animals

Freshwater aquatic species exposed to low concentrations of TCDD (in the parts per trillion range) for 4 days displayed toxic signs and died from 40 to 140 days later. Acute toxic effects were not noted in many of the aquatic species at the level of TCDD water solubility, 0.2 μg/liter. Horses exposed to TCDD in contaminated waste oil used to control dust in corrals, became sick and died.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 1.0 μg/liter
Chronic toxicity: <0.001 μg/liter

Saltwater

No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of TCDD in water are:

Risk	Concentration
10^{-5}	.00013 ng/liter
10^{-6}	.000013 ng/liter
10^{-7}	.0000013 ng/liter

CAG Unit Risk (USEPA): 1.6×10^{5} $(mg/kg/day)^{-1}$

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1,1,2,2-TETRACHLOROETHANE

Summary

1,1,2,2-Tetrachloroethane induces liver tumors when administered orally to mice, and it was shown to be mutagenic using microbial assays. Upon administration to pregnant mice, it reportedly had embryotoxic effects and increased the incidence of malformations. In experimental animals, acute and chronic exposure damages the liver, central nervous system, and kidneys. In humans, acute exposure depresses the central nervous system and may be fatal. Chronic effects in humans include liver damage, gastrointestinal disturbances, and effects on the central nervous system.

CAS Number: 79-34-5

Chemical Formula: $C_2H_2Cl_4$

IUPAC Name: 1,1,2,2-Tetrachloroethane

Important Synonyms and Trade Names: sym-Tetrachloroethane, acetylene tetrachloride, dichloro-2,2-dichloroethane

Chemical and Physical Properties

Molecular Weight: 167.85

Boiling Point: 146.2°C

Melting Point: -36°C

Specific Gravity: 1.5953 at 20°C

Solubility in Water: 2,900 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, acetone, benzene, petroleum ether, carbon tetrachloride, chloroform, carbon disulfide, dimethylformamide, and oils

Log Octanol/Water Partition Coefficient: 2.56

Vapor Pressure: 5 mm Hg at 20°C

Vapor Density: 5.79

Transport and Fate

Relatively little information is available pertaining specifically to the environmental transport and fate of 1,1,2,2-tetrachloroethane. However, predictions concerning these processes can be made based on comparison with similar compounds such as 1,1,1-trichloroethane. Photolysis and oxidation do not appear to be significant aquatic fate processes for 1,1,2,2-tetrachloroethane. However, based on analogy with 1,1,1-trichloroethane, stratospheric photodissociation by high energy ultraviolet light and tropospheric photooxidation via reaction with hydroxyl radicals seem likely to be relatively important fates. No information related specifically to hydrolysis of 1,1,2,2-tetrachloroethane in the environment is available. However, the low observed reactivity of 1,1,1-trichloroethane suggests that hydrolysis of 1,1,2,2-tetrachloroethane would occur too slowly to be an important fate process. Available data indicate that relatively rapid volatilization of 1,1,2,2-tetrachloroethane from surface waters can occur. Thus, although some of this compound will be absorbed from the atmosphere by surface water and return to earth in precipitation, atmospheric photooxidation and photodissociation are probably the most important environmental fates.

Based on analogy with 1,1,1-trichloroethane, sorption of 1,1,2,2-tetrachloroethane to clay sediments probably is not an important process. The log octanol/water partition coefficient of 2.56 for this compound indicates that sorption by organic particulates and bioaccumulation may occur to some extent; however, no adequate empirical data are available. Available information concerning related compounds suggests that biotransformation and biodegradation occur at low rates or not at all.

Health Effects

1,1,2,2-Tetrachloroethane is a liver carcinogen when administered orally to mice. IARC concludes that there is limited evidence for its carcinogenicity in experimental animals. This compound is mutagenic in at least two bacterial tester strains. Administration of 300-400 mg/kg/day to mice during organogenesis is reported to produce embryotoxic effects and slightly increase the incidence of malformations.

1,1,2,2-Tetrachloroethane produces acute and chronic toxic effects in laboratory animals exposed by various routes. Toxic action is primarily on the liver. However, effects on the central nervous system, kidneys, and other tissues are also reported; and acute exposure can be fatal. The oral LD_{50} in rats is 250 mg/kg.

Numerous deaths in humans have been reported, primarily as a result of occupational exposure by ingestion, inhalation, or skin contact. Acute exposure produces central nervous system depression. Chronic effects include hepatotoxicity and gastrointestinal disturbances in addition to central nervous system effects such as tremors, dizziness, headache, paralysis, and polyneuritis.

Toxicity to Wildlife and Domestic Animals

Acute values for freshwater species range from 9,320 µg/liter for an invertebrate species to approximately 20,000 µg/liter for two species of fish. An embryo-larval test conducted with the fathead minnow provides a chronic value of 2,400 µg/liter and an acute-chronic ratio of 8.5 for this species. Among saltwater species, acute values of 9,020 µg/liter for the mysid shrimp and 12,300 µg/liter for the sheepshead minnow are reported. Exposure to 1,1,2,2-tetrachloroethane affects chlorophyll a and cell numbers of algae exposed to approximately 141,000 µg/liter in a freshwater species and 6,300 µg/liter in a saltwater species. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 5.0.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of 1,1,2,2-tetrachloroethane in water are:

Risk	Concentration
10^{-5}	1.7 µg/liter
10^{-6}	0.17 µg/liter
10^{-7}	0.017 µg/liter

CAG Unit Risk (USEPA): 0.2 $(mg/kg/day)^{-1}$

NIOSH Recommended Standard: 7 mg/m^3 TWA

OSHA Standard (skin): 35 mg/m^3

ACGIH Threshold Limit Values (skin): 7 mg/m^3 TWA
35 mg/m^3 STEL

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TETRACHLOROETHYLENE

Summary

Tetrachloroethylene (PCE, perchloroethylene) induced liver tumors when administered orally to mice and was found to be mutagenic using a microbial assay system. Reproduction toxicity was observed in pregnant rats and mice exposed to high concentrations. Animals exposed by inhalation to tetrachloroethylene exhibited liver, kidney, and central nervous system damage.

CAS Number: 127-18-4

Chemical Formula: C_2Cl_4

IUPAC Name: Tetrachloroethene

Important Synonyms and Trade Names: Perchloroethylene, PCE

Chemical and Physical Properties

Molecular Weight: 165.83

Boiling Point: 121°C

Melting Point: -22.7°C

Specific Gravity: 1.63

Solubility in Water: 150 to 200 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, and benzene

Log Octanol/Water Partition Coefficient: 2.88

Vapor Pressure: 14 mm Hg at 20°C

Transport and Fate

Tetrachloroethylene (PCE) rapidly volatiziles into the atmosphere where it reacts with hydroxyl radicals to produce HCl, CO, CO_2 and carboxylic acid. This is probably the most important transport and fate process for tetrachloroethylene in the environment. PCE will leach into the groundwater, especially in soils of low organic content. In soils with high levels of organics, PCE adsorbs to these materials and can

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be bioaccumulated to some degree. However, it is unclear if tetrachloroethylene bound to organic material can be degraded by microorganisms or must be desorbed to be destroyed. There is some evidence that higher organisms can metabolize PCE.

Health Effects

Tetrachloroethylene was found to produce liver cancer in male and female mice when administered orally by gavage (NCI 1977). Unpublished gavage studies in rats and mice performed by the National Toxicology Program (NTP) showed hepatocellular carcinomas in mice and a slight, statistically insignificant increase in a rare type of kidney tumor.[1] NTP is also conducting an inhalation carcinogenicity study. Elevated mutagenic activity was found in Salmonella strains treated with tetrachloroethylene. Delayed ossification of skull bones and sternebrae were reported in offspring of pregnant mice exposed to 2,000 mg/m^3 of tetrachloroethylene for 7 hours/day on days 6-15 of gestation. Increased fetal resorptions were observed after exposure of pregnant rats to tetrachloroethylene. Renal toxicity and hepatotoxicity have been noted following chronic inhalation exposure of rats to tetrachloroethylene levels of 1,356 mg/m^3. During the first 2 weeks of a subchronic inhalation study, exposure to concentrations of 1,622 ppm (10,867 mg/m^3) of tetrachloroethylene produced signs of central nervous system depression, and cholinergic stimulation was observed among rabbits, monkeys, rats, and guinea pigs.

Toxicity to Wildlife and Domestic Animals

Tetrachloroethylene is the most toxic of the chloroethylenes to aquatic organisms but is only moderately toxic relative to other types of compounds. The limited acute toxicity data indicate that the LC_{50} value for saltwater and freshwater species are similar, around 10,000 µg/liter; the trout was the most sensitive (LC_{50} = 4,800 µg/liter). Chronic values were 840 and 450 µg/liter for freshwater and saltwater species, respectively, and an acute-chronic ratio of 19 was calculated.

No information on the toxicity of tetrachloroethylene to terrestrial wildlife or domestic animals was available in the literature reviewed.

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[1] J. Mennear, NTP Chemical Manager; personal communication, 1984.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic to aquatic organisms.

Freshwater

Acute toxicity: 5,280 µg/liter
Chronic toxicity: 840 µg/liter

Saltwater

Acute toxicity: 10,200 µg/liter
Chronic toxicity: 450 µg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of tetrachloroethylene in water are:

Risk	Concentration
10^{-5}	8.0 µg/liter
10^{-6}	0.8 µg/liter
10^{-7}	0.08 µg/liter

CAG Unit Risk (USEPA): 5.1×10^{-2} $(mg/kg/day)^{-1}$

NIOSH Recommended Standards (air): 335 mg/m^3 TWA
670 mg/m^3 15-min Ceiling Level

OSHA Standards (air): 670 mg/m^3 TWA
1,340 mg/m^3 Ceiling Level
2,010 mg/m^3 for 5 min every 3 hr, Peak Level

REFERENCES

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TETRAETHYL LEAD

Summary

Tetraethyl lead (TEL) is likely to have adverse effects on human reproduction and embryonic development. Human exposure has been associated with adverse effects on the central nervous system, peripheral nerves, kidneys, and hematopoietic system.

CAS Number: 78-00-2

Chemical Formula: $(C_2H_5)_4Pb$

IUPAC Name: Tetraethyl lead

Important Synonyms and Trade Names: Tetraethyl plumbane, TEL

Chemical and Physical Properties

Molecular Weight: 323.45

Boiling Point: Approximately 200°C

Melting Point: -136.8°C

Specific Gravity: 1.653 at 20°C

Solubility in Water: Insoluble

Solubility in Organics: Soluble in most organic solvents

Vapor Pressure: 1 mm Hg at 38.4°C

Flash Point: 77°C

Transport and Fate

Volatilization of tetraethyl lead (TEL) from aquatic or terrestrial systems may be a significant transport process. However, its presence in the atmosphere probably would be transient, because photochemical decomposition occurs readily. The organic and inorganic lead compounds formed can then be removed from the atmosphere by wet or dry deposition.

Sorption processes in sediments and soils may be important for TEL. However, TEL is generally not stable in aerobic environ-

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ments. TEL decomposes slowly at room temperature and more rapidly at elevated temperatures. In aquatic systems, a significant portion of TEL is probably oxidized in the water column. Overall, most TEL probably undergoes conversion to inorganic lead compounds relatively quickly. The types of compounds formed and their subsequent environmental fates are determined by local physical and chemical conditions.

Health Effects

There are no reports of carcinogenicity, mutagenicity, or teratogenicity in humans as a result of exposure to TEL. Young female Swiss mice developed malignant lymphomas after subcutaneous exposure to TEL. However, the significance of these findings could not be evaluated because of the low tumor incidence in only one sex and because the type of tumor observed occurs spontaneously and with variable incidence in the strain of mouse studied. Although specific results with TEL are not available, lead is reported to have adverse effects on human reproduction and embryonic, fetal, and postnatal development. Fetotoxicity and postimplantation mortality are reported to occur after oral administration to pregnant rats.

TEL is toxic to humans and experimental animals by oral, inhalation, and cutaneous routes of exposure. Effects are commonly seen in the central nervous system, peripheral nerves, the kidney, and hematopoietic system. In humans, TEL intoxication is reportedly characterized by insomnia, hallucinations, emotional instability, and increased physical activity of an erratic nature. After exposure to high concentrations, coma and death may occur. An oral LD_{LO} value of 17 mg/kg and an inhalation LC_{50} value of 850 mg/m^3 for 60 minutes are reported for the rat. Lethal dermal doses of 547 mg/kg and 830 mg/kg are reported for the dog and rabbit, respectively.

Toxicity to Wildlife and Domestic Animals

Although lead is known to occur in the tissues of many free-living wild animals, including birds, mammals, fishes, and invertebrates, reports of poisoning usually involve waterfowl that ingest lead shot. There also is evidence that lead, at concentrations occasionally found near roadsides and smelters, can eliminate or reduce populations of bacteria and fungi on leaf surfaces and in soil. Cases of lead poisoning have been reported for a variety of domestic animals, including cattle, horses, dogs, and cats. It is probable that poisoning in wildlife and domestic animals involves exposure to elemental lead or lead compounds other than TEL. One study reports an EC_{50} value of 150 μg/liter in a saltwater alga exposed to TEL.

Regulations and Standards

NIOSH Recommended Standard: 0.10 mg/m^3 TWA (as Pb)

OSHA Standard: 0.075 mg/m^3 (as Pb)

ACGIH Threshold Limit Values: 0.1 mg/m^3 TWA (as Pb)
0.3 mg/m^3 STEL (as Pb)

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TETRAHYDROFURAN

Summary

Tetrahydrofuran was found to be mutagenic using a microbial assay system and caused chromosomal aberrations in Chinese Hamster Ovary Cells. Technical tetrahydrofuran has been shown to irritate the skin and cause liver and kidney damage.

CAS Number: 109-99-9

Chemical Formula: C_4H_8O

IUPAC Name: Tetrahydrofuran

Important Synonyms and Trade Names: Diethylene oxide, butylene oxide, tetramethylene oxide, hydrofuran, THF

Chemical and Physical Properties

Molecular Weight: 72.12

Boiling Point: 67°C

Melting Point: -65°C

Specific Gravity: 0.8892 at 20°C

Solubility in Water: Soluble in water

Solubility in Organics: Very soluble in alcohol, ether, acetone, benzene, and other organic solvents

Vapor Pressure: 143 mm Hg at 20°C

Vapor Density: 2.5

Transport and Fate

Little information is available on the transport and fate of tetrahydrofuran in the natural environment. It has a relatively high vapor pressure and should therefore volatilize into the atmosphere. Upon exposure to ultraviolet radiation, it produces ozone, aldehydes, and epoxides and apparently is not very persistent. In aquatic systems, its volatilization would be somewhat limited by its rather high water solubility. Its water solubility also suggests that tetrahydrofuran probably

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moves readily through soil and is not bioaccumulated to any substantial degree.

Health Effects

Tetrahydrofuran is currently being tested by the Nation Toxicology Program to assess its carcinogenic potential. No evidence of carcinogenic, reproductive, or teratogenic effec associated with exposure to tetrahydrofuran was found in the literature reviewed. It was found to be mutagenic in a mic assay on *Escherichia coli* and caused chromosomal aberrations but not sister chromated exchange in Chinese hamster ovary cells.

Exposure to 590 mg/m^3 tetrahydrofuran for 6 hours/day caused decreased pulse pressure in dogs after 4 weeks, but no histopathological changes occurred in major organs after 12 weeks. Daily (oral?) administration of 20 mg/kg for 6 mor caused weight loss, paralysis of the hind limbs, hyperemia of the viscera and protein dystrophy of the liver (Pozdnyakov in USEPA 1980). Pure tetrahydrofuran does not appear to caus toxic effects even at very high concentrations (greater than 10,000 g/m^3), but the technical compound, which is contaminat with peroxides, causes skin irritation and liver and kidney damage. The oral LD$_{50}$ in rats was reported to be 2,800 mg/kg

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of tetrahydrofuran to life and domestic animals was available in the literature rev

Regulations and Standards

OSHA Standard (air): 590 mg/m^3 TWA

ACGIH Threshold Limit Values: 590 mg/m^3 TWA
735 mg/m^3 STEL

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THALLIUM

Summary

Acute exposure to soluble thallium compounds has been associated in humans with gastrointestinal irritation; damage of the liver, kidneys, and central and peripheral nervous systems; pulmonary edema; degenerative changes in the adrenals; and ocular effects.

CAS Number: 7440-28-0

Chemical Formula: Tl

IUPAC Name: Thallium

Chemical and Physical Properties

Atomic Weight: 204.37

Boiling Point: 1,457°C

Melting Point: 303.5°C

Specific Gravity: 11.85

Solubility in Water: Insoluble (many compounds are soluble)

Transport and Fate

In reducing environments, thallium may be precipitated as the metal or as thallium sulfide. However, much of the thallium present in aquatic systems is likely to remain in solution and be transported to the oceans. Active removal of some dissolved thallium by sorption to clay minerals and hydrous metal oxides present in bed sediments is probably an important environmental fate process. Thallium is readily taken up by aquatic organisms, and bioaccumulation may also be an important fate process. Results of limited studies with algae suggest that thallium may also be available for food chain magnification. There is no evidence to suggest that photolysis or volatilization are important environmental processes. Although there is speculation that thallium can be methylated under aerobic conditions by electrophilic attack, biotransformation does not appear to be an important process in aquatic systems.

Health Effects

There is no evidence that thallium is carcinogenic in humans or experimental animals, and it does not appear to have significant mutagenic activity. Exposure to thallium salts during critical developmental stages is reported to produce achondioplasia in chickens and rats. No other significant teratogenic effects are reported.

Thallium, in the form of soluble compounds, is readily absorbed through the skin and gastrointestinal tract. Symptoms associated with acute poisoning in humans include gastrointestinal irritation; liver and kidney damage; pulmonary edema; degenerative changes in the adrenals, peripheral nervous system, and central nervous system; and ocular effects, including optic neuritis and, rarely, cataracts. The estimated lethal dose for humans is 8 to 12 mg/kg. In experimental animals, thallium compounds produce effects similar to those seen in humans. Rats appear to be particularly sensitive to the cataractogenic activity of thallium. Regardless of the specific thallium compound tested, rate of intake, or route of administration, LD_{50} values for a variety of species range from about 3 to 92 mg/kg.

Toxicity to Wildlife and Domestic Animals

Acute and chronic toxicity of thallium to freshwater aquatic life occurs at concentrations as low as 1,400 and 40 µg/liter, respectively. Acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,130 µg/liter. Toxic effects would be expected to occur at lower concentrations among species more sensitive than those tested. Bioconcentration factors ranged from about 11 for the mussel *Mytilus edulis* to about 1.5×10^5 for other freshwater and marine invertebrates. Values of about 1×10^5 are reported for marine and freshwater fish.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 13 µg/liter

OSHA Standard: 100 µg/m^3 TWA

ACGIH Threshold Level Value: 0.1 mg/m^3 TWA (soluble compounds, as Tl)

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TITANIUM

Summary

Titanium dioxide produced injection-site tumors in rats given intramuscular injections and lung tumors in rats exposed by inhalation. In humans, inhalation exposure to high concentrations of titanium dioxide has caused slight fibrosis of the lung.

Background Information

Titanium is the ninth most abundant element in the crust of the earth and is almost always present in igneous rocks and in the sediments derived from them. Titanium is an active metal, but it resists decomposition because of the formation of a protective titanium dioxide film. The film is insoluble, repairable, and provides excellent corrosion resistance. Consequently, titanium resists corrosion in all naturally occurring environments, including air, soil, and water.

CAS Number: 7440-32-6

Chemical Formula: Ti

IUPAC Name: Titanium

Chemical and Physical Properties

Atomic Weight: 47.90

Boiling Point: 3,287°C

Melting Point: 1,660°C

Specific Gravity: 4.5

Solubility in Water: Insoluble

Transport and Fate

Most common titanium compounds are insoluble in water and partitioning of much of the titanium in aquatic systems into the bottom sediments would be expected. Atmospheric transport of titanium, and subsequent wet and dry deposition, can occur.

Health Effects

There is no evidence to suggest that titanium is carcinogenic in humans. Titanium dioxide is reported to have tumorigenic effects at the site of intramuscular injection in rats. Preliminary results of a 2-year E.I. Dupont (1983) study show increased incidences of squamous cell carcinoma of the lung and bronchioalveolar adenoma in rats exposed to airborne concentrations of 250 mg/m^3 of titanium dioxide. Results of a 2-year feeding study conducted for the National Cancer Institute indicated that titanium dioxide was not carcinogenic in rats and mice under the conditions of the bioassay. There are no reports of mutagenic, teratogenic, or reproductive effects associated with exposure to titanium in humans or experimental animals.

In humans, most of the body burden of titanium is in the lungs. About one-third of inhaled titanium is thought to be retained in the lungs. Slight fibrosis has been observed after inhalation exposure to high concentrations of titanium dioxide dust. However, titanium generally acts as an inert particulate material in the lungs. Titanium does not appear to produce significant skin irritation or to cause toxic effects after ingestion in humans or experimental animals.

Toxicity to Wildlife and Domestic Animals

The available data are not adequate to characterize the toxicity of titianium to wildlife and domestic animals.

Regulations and Standards

ACGIH Threshold Limit Values:

10 mg/m^3 TWA (titanium dioxide, total dust)
5 mg/m^3 TWA (titanium dioxide, respirable dust)
20 mg/m^3 STEL (titanium dioxide)

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TOLUENE

Summary

Toluene has been shown to be embryotoxic in experimental animals, and the incidence of cleft palate increased in the offspring of dosed mice. Chronic inhalation exposure to high levels of toluene caused cerebellar degeneration and an irreversible encephalopathy in animals. In humans, acute exposure depressed the central nervous system and caused narcosis.

CAS Number: 108-88-3

Chemical Formula: $C_6H_5CH_3$

IUPAC Name: Methylbenzene

Important Synonyms and Trade Names: Toluol, phenylmethane

Chemical and Physical Properties

Molecular Weight: 92.13

Boiling Point: 110.6°C

Melting Point: -95°C

Specific Gravity: 0.8669 at 20°C

Solubility in Water: 534.8 mg/liter

Solubility in Organics: Soluble in acetone, ligroin, and carbon disulfide; miscible with alcohol, ether, benzene, chloroform, glacial acetic acid, and other organic solvents

Log Octanol/Water Partition Coefficient: 2.69

Vapor Pressure: 28.7 mm Hg at 25°C

Vapor Density: 3.14

Flash Point: 4.4°C

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Transport and Fate

Volatilization appears to be the major route of removal of toluene from aquatic environments, and atmospheric reaction of toluene probably subordinate all other fate processes (USE 1979). Photooxidation is the primary atmospheric fate proces for toluene, and benzaldehyde is reported to be the principal organic product. Subsequent precipitation or dry deposition can deposit toluene and its oxidation products into aquatic and terrestrial systems. Direct photolytic cleavage of toluen is energetically improbable in the troposphere, and oxidation and hydrolysis are probably not important as aquatic fates.

The log octanol/water partition coefficient of toluene indicates that sorption processes may be significant. However, no specific environmental sorption studies are available, and the extent to which adsorption by sedimentary and suspended organic material may interfere with volatilization is unknown. Bioaccumulation is probably not an important environmental fate process. Although toluene is known to be degraded by microorganisms and can be detoxified and excreted by mammals, the available data do not allow estimation of the relative importance of biodegradation/biotransformation processes. Almost all toluene discharged to the environment by industry is in the form of atmospheric emissions.

Health Effects

There is no conclusive evidence that toluene is carcinogenic or mutagenic in animals or humans (USEPA 1980). The National Toxicological Program is currently conducting an inhalation carcinogenicity bioassay in rats and mice.

Oral administration of toluene at doses as low as 260 mg/k produced a significant increase in embryonic lethality in mice (USEPA 1980). Decreased fetal weight was observed at doses as low as 434 mg/kg, and an increased incidence of cleft palate was seen at doses as low as 867 mg/kg. However, other research have reported that toluene is embryotoxic but not teratogenic in laboratory animals. There are no accounts of a teratogenic effect in humans after exposure to toluene.

Acute exposure to toluene at concentrations of 375-1,500 m produces central nervous system depression and narcosis in humans (ACGIH 1980). However, even exposure to quantities sufficient to produce unconsciousness fail to produce residual organ damage. The rat oral LD_{50} value and inhalation LC_{LO} value are 5,000 mg/kg and 15,000 mg/m^3, respectively. Chronic inhalation exposure to toluene at relatively high concentration produces cerebellar degeneration and an irreversible encephalop in mammals.

Toluene in sufficient amounts appears to have the potential to alter significantly the metabolism and resulting bioactivity of certain chemicals. For example, coadministration of toluene along with benzene or styrene has been shown to suppress the metabolism of benzene or styrene in rats.

Toxicity to Wildlife and Domestic Animals

Of five freshwater species tested with toluene, the cladoceran *Daphnia magna* was most resistant to any acute effects (USEPA 1980). The EC_{50} and LC_{50} values for all five species range from 12,700 to 313,000 μg/liter. No chronic tests are available for freshwater species. The two freshwater algal species tested are relatively insensitive to toluene with EC_{50} values of 245,000 μg/liter or greater being reported. For saltwater species, EC_{50} and LC_{50} values range from 3,700 μg/liter for the bay shrimp to 1,050 mg/liter for the Pacific oyster. The chronic value in an embryo-larval test for the sheepshead minnow is reported to be between 3,200 and 7,700 μg/liter, and the acute-chronic ratio is between 55 and 97. In several saltwater algal species and kelp, effects occur at toluene concentrations from 8,000 to more than 433,000 μg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest concentrations of toluene known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 17,500 μg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 6,300 μg/liter
Chronic toxicity: 5,000 μg/liter

Human Health

Criterion: 14.3 mg/liter

NIOSH Recommended Standards: 375 mg/m^3 TWA
560 mg/m^3 STEL

OSHA Standards: 750 mg/m^3 TWA
1,120 mg/m^3 Ceiling Level

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TOXAPHENE

Summary

Toxaphene is a chlorinated organic pesticide and is persistent in the natural environment. In animal bioassays, it induced liver cancer in mice and thyroid tumors in rats. Toxaphene is fetotoxic and decreases spermatogenesis. Chronic exposure to toxaphene has been shown to damage the liver and kidneys and stimulate the central nervous system in animals. In humans, symptoms of acute intoxication include vomiting, convulsions, cyanosis, and coma. Toxaphene is highly toxic to aquatic organisms.

Background Information

Toxaphene consists primarily of chlorinated camphene and a mixture of related compounds and isomers. In general, the physical and chemical properties reported below are average values.

CAS Number: 8001-35-2

Chemical Formula: $C_{10}H_{10}Cl_8$ (average formula)

Important Synonyms and Trade Names: Camphechlor, chlorinated camphene, Attac, Phenacide, Strobane-T

Chemical and Physical Properties

Molecular Weight: 414

Boiling Point: Greater than 120°C

Melting Point: 65-95°C

Specific Gravity: 1.64 at 25°C

Solubility in Water: 0.4 to 3.0 mg/liter

Solubility in Organics: Very soluble in most organic solvents

Log Octanol/Water Partition Coefficient: 3.3

Vapor Pressure: 0.2 to 0.4 mm Hg at 25°C

Flash Point: 135°C (closed cup)

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Transport and Fate

Because toxaphene is a complex mixture of polychlorinated camphene derivatives, an inclusive assessment of its environmental transport and fate is difficult. Photolysis, oxidation, and hydrolysis do not appear to be important fate processes in aquatic systems. It is persistent in the environment, and transport through soil, water, and air can occur relatively easily. Although little information is available, it appears that volatilization may be an important transport process, especially for the higher chlorinated bornane structures with very low solubility in water. Toxaphene is very stable to biological and chemical degradation processes in aerobic environmental systems, but it does undergo partial reduction (loss of chloride content) in anaerobic environments. Accordingly, although biodegradation can occur, it depends on transport of toxaphene to anaerobic environments. A dominant process in aquatic systems is direct sorption on sediments, or adsorption onto particulates, followed by deposition into sediment where biological and chemical reduction may occur. The rate of loss of toxaphene from aquatic systems is partially determined by particulate loading and quality of the water body. The physical and chemical properties of the individual toxaphene components determine which compounds will be sorbed and subsequently reduced. Bioaccumulation is an important environmental process for toxaphene. Adsorption by biota is rapid, and significant uptake can occur in natural systems.

Health Effects

The results of a bioassay conducted for the Carcinogenesis Testing Program of the National Cancer Institute indicate that toxaphene causes increased incidences of hepatocellular carcinomas in mice, and suggest that it is carcinogenic for the thyroid in the rat (NCI 1979). IARC has concluded that toxaphene is an animal carcinogen and a suspected human carcinogen. Toxaphene has produced both positive and negative results in a series of different mutagenicity assays. Studies concerning the reproductive effects of toxaphene suggest that oral administration may produce maternal and fetal toxicity. However, toxaphene does not appear to have teratogenic effects.

Acute exposure to toxaphene causes effects due primarily to central nervous system stimulation. Subchronic exposure results in kidney changes, as well as changes in blood chemistry. Symptoms of acute oral toxaphene intoxication in humans include vomiting, convulsions, cyanosis, and coma. A minimum lethal oral dose of 40 mg/kg is reported for humans. In rats, pathological effects of toxaphene include cloudy swelling and congestion of the kidneys, fatty degeneration and necrosis of the liver, and decreased spermatogenesis. Toxaphene in the diet is reported to inhibit hepatobiliary function in rats. An oral LD_{50} value of 40 mg/kg is reported for the rat. Although there also are reports of toxaphene toxicity due to dermal and inhalation exposure in humans and experimental animals, most available information concerns effects due to ingestion.

Toxicity to Wildlife and Domestic Animals

Mean acute values for freshwater invertebrate species range from 1.3 μg/liter for the stonefly to 180 μg/liter for the midge. Values for fish species range from 2 μg/liter for largemouth bass to 20 μg/liter for the guppy. Mean species chronic values range from 0.037 μg/liter for the fathead minnow to 1.8 μg/liter for the midge. Freshwater acute-chronic ratios range from 71 to 265. Species mean acute values for saltwater invertebrates range from 0.11 μg/liter for a copepod to 1,120 μg/liter for the hard shell clam. Values for fish species range from 0.5 μg/liter for the pinfish to 8.2 μg/liter for the three-spine stickleback. A chronic value of 1.66 μg/liter is reported for the sheepshead minnow. Bioconcentration factors among aquatic organisms range from about 1,200 to more than 50,000. Toxaphene concentrations of from 0.15 to 1,000 μg/liter are reported to cause deleterious effects in aquatic plant species.

Toxaphene has a relatively high degree of toxicity in aquatic organisms and has resulted in fish kills and adverse effects on fish development and reproduction. Although toxaphene is relatively less toxic to birds and mammals, bioaccumulation may result in exposure to excessive concentrations. Bird kills due to toxaphene have been reported.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 1.6 μg/liter
Chronic toxicity: 0.013 μg/liter

Saltwater

Acute toxicity: 0.070 μg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of toxaphene in water are:

Risk	Concentration
10^{-5}	7.1 ng/liter
10^{-6}	0.71 ng/liter
10^{-7}	0.07 ng/liter

CAG Unit Risk (USEPA): 1.13 $(mg/kg/day)^{-1}$

OSHA Standard: 0.5 mg/m^3 TWA

ACGIH Threshold Limit Values: 0.5 mg/m^3 TWA
1 mg/m^3 STEL

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TRICHLOROBENZENE

Summary

High doses of trichlorobenzene (TCB) have been shown to be embryotoxic to the offspring of exposed rats. Dermal applications of TCB increased the incidence of amyloidosis in a number of organs in mice and consequently shortened the animals' lifespans. Inhalation exposure to trichlorobenzene had minor effects on the liver and kidneys in several species of experimental animals; in a study in mice, it also damaged the bone marrow.

CAS Number: 1,2,3-TCB: 87-61-6
1,2,4-TCB: 120-82-1
1,3,5-TCB: 108-70-3

Chemical Formula: $C_6H_3Cl_3$

IUPAC Names: 1,2,3-Trichlorobenzene; 1,2,4-Trichlorobenzene; 1,3,5-Trichlorobenzene

Important Synonyms and Trade Names: Trichlorobenzene, TCB

Chemical and Physical Properties

Molecular Weight: 181.45

Boiling Point: 1,2,3-TCB: 219°C
1,2,4-TCB: 213°C
1,3,5-TCB: 208°C

Melting Point: 1,2,3-TCB: 54°C
1,2,4-TCB: 17°C
1,3,5-TCB: 64°C

Specific Gravity: 1,2,4-TCB: 1.4542

Solubility in Water: 1,2,4-TCB: 30 mg/liter at 25°C

Solubility in Organics: Sparingly soluble in alcohol; freely soluble in benzene and carbon disulfide

Log Octanol/Water Partition Coefficient: 1,2,3-TCB: 4.1
1,2,4-TCB: 4.3 (calculate

Vapor Pressure: Approximately 0.4 mm Hg at 25°C

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Transport and Fate

There is little information on the transport and fate of trichlorobenzenes, and what is available primarily concerns 1,2,4-trichlorobenzene (1,2,4-TCB). Although there is no inform tion on the sorption of 1,2,4-TCB to soils and sediments, the high log octanol/water partition coefficient suggests that this compound would be adsorbed to organic materials in soil and sediment. The volatility of 1,2,4-TCB is relatively low, but it has been found to volatilize readily from aerated and quiescent waters, with a half-life of less than 1 hour and 4-7 hours in each medium, respectively. Thus, air transport is also likely. Sorption to suspended solids does, however, reduce the rate of volatilization.

1,2,4-TCB has been shown to be oxidized in the atmosphere via attack by hydroxyl radicals. It is not known if the compoun is broken down through photolysis or hydrolysis. Biodegradation of 1,2,4-TCB has been shown to occur in waste treatment studies. However, in the environment biodegradation is expected to be slower.

Health Effects

There are no reports indicating carcinogenic, teratogenic, or mutagenic activity of the trichlorobenzenes in humans or animals. No specific reproductive effects have been found for the TCBs, but embryotoxicity has been noted at a dose leve that produces maternal toxicity in rats (Kitchin and Ebron 1983).

Several animal studies on the subchronic toxicity of trichlorobenzenes have been reported. Inhalation studies with 1,2,4-TCB of 1.5 to 6 months duration in rats, rabbits, dogs, and monkeys have not shown major irreversible effects, although some effects on liver and kidney were found (transient histological changes and increased relative liver weight; Kociba et al. 1981, Coate et al. 1982). Increased urinary porphyrin levels were also noted (Kociba et al. 1981). Zub (1978) reporte that mice exposed to TCB (isomers unspecified) for 3 weeks to 3 months showed indications of bone marrow damage. In a chronic study in which mice were administered 1,2,4-TCB by dermal application, there was a treatment-related increase in the incidence of amyloidosis, which affected a number of organs and was considered a primary cause of death (Yamamoto et al. 1982).

TCB is an inducer of the microsomal mixed function oxidases and therefore will increase metabolism, leading to the inactivation or activation of chemicals affected by this system.

Toxicity to Wildlife and Domestic Animals

Only 1,2,4-TCB has been studied for its toxic effect on aquatic wildlife. Acute LC_{50} values for the freshwater species *Daphnia magna*, rainbow trout, and fathead minnow are 50.2, 1.5, and 2.87 mg/liter, respectively. In the saltwater species, the LC_{50} values are 0.45 and 21.4 mg/liter for mysid shrimp and sheepshead minnow, respectively. Chronic toxicity in the early life stage of the fathead minnow occurred at concentrations of 1,2,4-TCB that ranged from 0.206 to 0.705 mg/liter. In freshwater and saltwater algae, the EC_{50} values for 1,2,4-TCB on chlorophyll are 35.3 and 8.75, respectively; and for its effect on cell numbers, the EC_{50} values are 36.7 and 8.93 mg/liter, respectively.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

The available data are not adequate for establishing criteria.

ACGIH Threshold Limit Value: 1,2,4-TCB: 40 mg/m^3 TWA

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2,3,6-TRICHLOROBENZOIC ACID

Summary

2,3,6-Trichlorobenzoic acid (2,3,6-TBA) was slightly irritating when applied to the shaved skin of mice.

CAS Number: 50-31-7

Chemical Formula: $C_7H_3Cl_3O_2$

IUPAC Name: 2,3,6-Trichlorobenzoic acid

Important Synonyms and Trade Names: Benzabar, Benzac, 2,3,6-TBA, and Trysben

Chemical and Physical Properties

Molecular Weight: 225.45

Melting Point: 125 to 126°C

Solubility in Water: 8,400 mg/liter

Solubility in Organics: Soluble in acetone, benzene, chloroform, ethanol, ethyl acetate, ethylene glycol, methanol, and xylene

Log Octanol/Water Partition Coefficient: 3.5 (calculated)

Vapor Pressure: Very low

Transport and Fate

2,3,6-Trichlorobenzoic acid (2,3,6-TBA) is rather persistent in the environment. It is not very volatile. It is fairly soluble in water and will leach through soils, although it has a rather high log octanol/water partition coefficient, which would suggest some adsorption to soil organics. 2,3,6-TBA is relatively resistant to photolysis and biodegrades slowly.

Health Effects

Only limited information on the toxicity of 2,3,6-TBA was available in the sources reviewed. No information was found on the carcinogenicity, mutagenicity, or reproductive toxicity of this compound. One subchronic study reported no histological changes in organs of rats exposed to doses of

750 mg/kg of the sodium salt of 2,3,6-TBA. However, the oral LD_{50} in rats was reported to be 650-1,000 mg/kg. 2,3,6-TBA was slightly irritating when applied to the shaved skin of mice.

Toxicity to Wildlife and Domestic Animals

The 48-hour LC_{50} values for the bluegill and the largemouth bass were 1,750 and 1,250 mg/liter, respectively. 2,3,6-TBA is a herbicide used primarily to control broadleaf plants. Therefore, these plants will be susceptible to its effects. No other information on the environmental toxicity of 2,3,6-TBA was available in the sources reviewed.

Regulations and Standards

No regulations or standards have been established for 2,3,6-trichlorobenzoic acid.

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1,1,1-TRICHLOROETHANE

Summary

Preliminary results suggest that 1,1,1-trichloroethane (1,1,1-TCA) induces liver tumors in female mice. It was shown to be mutagenic using the Ames assay, and it causes transformation in cultured rat embryo cells. Inhalation exposure to high concentrations of 1,1,1-TCA depressed the central nervous system; affected cardiovascular function; and damaged the lungs, liver, and kidneys in animals and humans. Irritation of the skin and mucous membranes has also been associated with human exposure to 1,1,1-trichloroethane.

CAS Number: 71-55-6

Chemical Formula: CH_3CCl_3

IUPAC Name: 1,1,1-Trichloroethane

Important Synonyms and Trade Names: Methyl chloroform, chlorothene, 1,1,1-TCA

Chemical and Physical Properties

Molecular Weight: 133.4

Boiling Point: 74.1°C

Melting Point: -30.4°C

Specific Gravity: 1.34 at 20°C (liquid)

Solubility in Water: 480-4,400 mg/liter at 20°C (several divergent values were reported in the literature)

Solubility in Organics: Soluble in acetone, benzene, carbon tetrachloride, methanol, ether, alcohol, and chlorinated solvents

Log Octanol/Water Partition Coefficient: 2.17

Vapor Pressure: 123 mm Hg at 20°C

Vapor Density: 4.63

Transport and Fate

1,1,1-Trichloroethane (1,1,1-TCA) disperses from surface water primarily by volatilization. Several studies have indicated that 1,1,1-trichloroethane may be adsorbed onto organic materials in the sediment, but this is probably not an important route of elimination from surface water. 1,1,1-Trichloroethane can be transported in the groundwater, but the speed of transport depends on the composition of the soil.

Photooxidation by reaction with hydroxyl radicals in the atmosphere is probably the principal fate process for this chemical.

Health Effects

1,1,1-Trichloroethane was retested for carcinogenicity because in a previous study by NCI (1977), early lethality precluded assessment of carcinogenicity. Preliminary results indicate that 1,1,1-TCA increased the incidence of combined hepatocellular carcinomas and adenomas in female mice when administered by gavage (NTP 1984). There is evidence that 1,1,1-trichloroethane is mutagenic in Salmonella typhimurium and causes transformation in cultured rat embryo cells (USEPA 1980). These data suggest that the chemical may be carcinogenic.

Other toxic effects of 1,1,1-TCA are seen only at concentrations well above those likely in an open environment. The most notable toxic effects of 1,1,1-trichloroethane in humans and animals are central nervous system depression, including anesthesia at very high concentrations and impairment of coordination, equilibrium, and judgment at lower concentrations (350 ppm and above); cardiovascular effects, including premature ventricular contractions, decreased blood pressure, and sensitization to epinephrine-induced arrhythmia; and adverse effects on the lungs, liver, and kidneys. Irritation of the skin and mucous membranes resulting from exposure to 1,1,1-trichloroethane has also been reported. The oral LD_{50} value of 1,1,1-trichloroethane in rats is about 11,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

The acute toxicity of 1,1,1-trichloroethane to aquatic species is rather low, with the LC_{50} concentration for the most sensitive species tested being 52.8 mg/l. No chronic toxicity studies have been done on 1,1,1-trichloroethane, but acute-chronic ratios for the other chlorinated ethanes ranged from 2.8 to 8.7. 1,1,1-Tricholoroethane was only slighty bioaccumulated with a steady-state bioconcentration factor of nine and an elimination half-life of two days.

No information on the toxicity of 1,1,1-trichloroethane to terrestrial wildlife or domestic animals was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report, the lowest values of the two trichloroethanes (1,1,1 and 1,1,2) known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 18 mg/liter
Chronic toxicity: 8.4 mg/liter

Saltwater

Acute toxicity: 31.2 mg/liter
Chronic toxicity: No available data

Human Health

Criterion: 18.4 mg/liter

NIOSH Recommended Standard: 350 ppm (1,910 mg/m^3)/15 min Ceiling Level

OSHA Standard: 350 ppm (1,910 mg/m^3) TWA

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1,1,2-TRICHLOROETHANE

Summary

1,1,2-Trichloroethane induced liver tumors and pheochromocytomas in mice. It caused liver and kidney damage in dogs.

CAS Number: 79-00-5

Chemical Formula: $CH_2ClCHCl_2$

IUPAC Name: 1,1,2-Trichloroethane

Important Synonyms and Trade Names: Vinyl trichloride, ethane trichloride

Chemical and Physical Properties

Molecular Weight: 133.41

Boiling Point: 133.8°C

Melting Point: -36.5°C

Specific Gravity: 1.4397 at 20°C

Solubility in Water: 4,500 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, and chloroform

Log Octanol/Water Partition Coefficient: 2.17

Vapor Pressure: 19 mm Hg at 20°C

Vapor Density: 4.63

Transport and Fate

Volatilization and subsequent photooxidation in the troposphere are probably the primary transport and fate processes for 1,1,2-trichloroethane. Some sorption, bioaccumulation, and biodegradation may occur, but these processes are probably not very important processes for trichloroethane transport or fate.

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1,1,2-Trichloroethane induced heptacellular carcinomas and pheochromocytoma of the adrenal gland in male and female mice but did not produce a significant increase in tumor incidence in male or female rats (NCI 1977). It was not mutagenic when tested using the Ames assay. No information was found concerning the reproductive toxicity or teratogenicity of 1,1,2-trichloroethane. No chronic studies were found on the toxicity of 1,1,2-trichloroethane but single doses as low as 400 mg/kg caused liver and kidney damage in dogs. The oral LD_{50} value for 1,1,2-trichloroethane in rats is 835 mg/kg.

Toxicity to Wildlife and Domestic Animals

The acute LC_{50} values for 1,1,2-trichloroethane for freshwater aquatic organisms ranged from 18,000 to 81,700 µg/liter. One chronic test was conducted; this indicated that the acute-chronic ratio for 1,1,2-trichloroethane was around 8.7. No information on the toxicity of 1,1,2-trichloroethane to saltwater species, terrestrial wildlife, or domestic animals was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not sufficient for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 18,000 µg/liter
Chronic toxicity: 9,400 µg/liter

Saltwater

Acute toxicity: No available data
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of 1,1,2-trichloroethane in water are:

Risk	Concentration
10^{-5}	6.0 μg/liter
10^{-6}	0.6 μg/liter
10^{-7}	0.06 μg/liter

CAG Unit Risk (USEPA): 5.7×10^{-2} $(mg/kg/day)^{-1}$

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TRICHLOROETHYLENE

Summary

Trichloroethylene (TCE) induced hepatocellular carcinomas in mice and was mutagenic when tested using several microbial assay systems. Chronic inhalation exposure to high concentrations caused liver, kidney, and neural damage and dermatological reactions in animals.

CAS Number: 79-01-6

Chemical Formula: C_2HCl_3

IUPAC Name: Trichloroethene

Important Synonyms and Trade Names: Trichloroethene, TCE, and ethylene trichloride

Chemical and Physical Properties

Molecular Weight: 131.5

Boiling Point: 87°C

Melting Point: -73°C

Specific Gravity: 1.4642 at 20°C

Solubility in Water: 1,000 mg/liter

Solubility in Organics: Soluble in alcohol, ether, acetone, and chloroform

Log Octanol/Water Partition Coefficient: 2.29

Vapor Pressure: 60 mm Hg at 20°C

Vapor Density: 4.53

Transport and Fate

Trichloroethylene (TCE) rapidly volatilizes into the atmosphere where it reacts with hydroxyl radicals to produce hydrochloric acid, carbon monoxide, carbon dioxide, and carboxylic acid. This is probably the most important transport and fate process for trichloroethylene in surface water and in the upper layer of soil. TCE adsorbs to organic materials and can be bioaccumulated to some degree. However, it is unclear whether trichloroethylene bound to organic material can be degraded by microorganisms or must be desorbed to be destroyed. There is some evidence that higher organisms can metabolize TCE. Trichloroethylene leaches into the groundwater fairly readily, and it is a common contaminant of groundwater around hazardous waste sites.

Health Effects

Trichloroethylene is carcinogenic to mice after oral administration, producing hepatocellular carcinomas (NCI 1976, NTP 1982). It was found to be mutagenic using several microbial assay systems. Trichloroethylene does not appear to cause reproductive toxicity or teratogenicity. TCE has been shown to cause renal toxicity, hepatotoxicity, neurotoxicity, and dermatological reactions in animals following chronic exposure to levels greater than 2,000 mg/m^3 for 6 months. Trichloroethylene has low acute toxicity; the acute oral LD_{50} value in several species ranged from 6,000 to 7,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

There was only limited data on the toxicity of trichloroethylene to aquatic organisms. The acute toxicity to freshwater species was similar in the three species tested, with LC_{50} values of about 50 mg/liter. No LC_{50} values were available for saltwater species. However, a dose of 2 mg/liter caused erratic swimming and loss of equilibrium in the grass shrimp. No chronic toxicity tests were reported.

No information on the toxicity of trichloroethylene to domestic animals or terrestrial wildlife was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Toxicity

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 45 mg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 2 mg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of trichloroethylene in water are:

Risk	Concentration
10^{-5}	27 μg/liter
10^{-6}	2.7 μg/liter
10^{-7}	0.27 μg/liter

CAG Unit Risk (USEPA): 1.1×10^{-2} $(mg/kg/day)^{-1}$

NIOSH Recommended Standards (air): 540 mg/m^3 TWA
760 mg/m^3 10-min Ceiling Level

OSHA Standards (air): 540 mg/m^3 TWA
1,075 mg/m^3/15-min Ceiling Level
1,620 mg/m^3 for 5 min every 3 hr, Peak Concentration

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TRICHLOROFLUOROMETHANE

Summary

Inhalation exposure to high concentrations of trichlorofluoromethane adversely affects the heart and lungs in humans and animals.

CAS Number: 75-69-4

Chemical Formula: CCl_3F

IUPAC Name: Fluorotrichloromethane

Important Synonyms and Trade Names: Freon-11, fluorocarbon 11

Chemical and Physical Properties

Molecular Weight: 137.37

Boiling Point: 23.82 °C

Melting Point: -111 °C

Specific Gravity: 1.467 at 25 °C

Solubility in Water: 1,100 mg/liter

Solubility in Organics: Soluble in alcohol, ether, and other organic solvents

Log Octanol/Water Partition Coefficient: 2.53

Vapor Pressure: 667.4 mm Hg at 20 °C

Vapor Density: 5.04

Transport and Fate

Though no specific data are available, the high vapor pressure, low solubility, and low boiling point of trichlorofluoromethane make it likely that volatilization into the atmosphere is the major transport process for removal of this compound from aqueous systems. Once in the troposphere, trichlorofluoromethane remains stable and eventually diffuses upward to the stratosphere or is carried back to earth by precipitation.

Trichlorofluoromethane that reaches the stratosphere is broken down by high energy, short wavelength ultraviolet light and this process is thought to be its primary environmental fate. Chlorine atoms released by such photodissociation processes are theorized by some researchers to serve as a catalyst in destruction of the stratospheric ozone layer.

Photolysis, oxidation, and hydrolysis do not appear to be significant environmental fate processes for trichlorofluoromethane in aquatic systems. The log octanol/water partition coefficient of trichlorofluoromethane indicates that adsorption onto sediments may occur. However, data concerning sorption processes are inconclusive.

Health Effects

Based on limited available information, trichlorofluoromethane does not appear to be carcinogenic in animals or humans. Results of a National Cancer Institute Carcinogenesis Bioassay using mice were negative. However, results for rats were considered inconclusive because inadequate numbers of rats survived long enough to be at risk from late-developing tumors. Although genotoxicity data are scant, trichlorofluoromethane exhibits no mutagenic activity in Salmonella tester strains. There are no available data on the teratogenicity or reproductive toxicity of trichlorofluoromethane.

In humans, trichlorofluoromethane toxicity generally involves the intentional or unintentional acute inhalation of high vapor concentrations. There are reports of severe intoxication and death under such circumstances. The cardiovascular and bronchopulmonary actions of trichlorofluoromethane are its two most important toxicological features and are thought to be mediated at least in part by metabolic products that bind to lipid and protein cell constituents and affect vital processes such as cellular oxidation.

The LC_{50} value for a 4-hour exposure with rats is 26,200 ppm. During exposure, sublethal doses caused rapid respiration with some mild hyperactivity, while lethal doses caused hyperactivity, tremors, inactivity, irregular respiration, and death within four hours. Laboratory animals periodically exposed at high concentrations for several days may exhibit biochemical changes consistent with slowing of cellular oxidation. Furthermore, studies with experimental animals suggest that inhalation exposure to high concentrations of trichlorofluoromethane may produce various cardiovascular and circulatory abnormalities. Both absorption and elimination are relatively rapid in humans and experimental animals.

Toxicity to Wildlife and Domestic Animals

Data concerning the toxicity of trichlorofluoromethane to wildlife and domestic animals are not available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 32.3 mg/liter (for protection against the noncarcinogenic effects of trichlorofluoromethane in ambient water)

OSHA Standard: 1,000 ppm (5,600 mg/m^3) Ceiling Level

ACGIH Threshold Limit Value: 1,000 ppm (5,600 mg/m^3) Ceiling Level

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

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2,4,5-TRICHLOROPHENOL

Summary

2,4,5-Trichlorophenol (2,4,5-TCP) promotes the formation of skin tumors in mice. In addition, although 2,4,5-trichlorophenol has not been tested in a complete carcinogenicity bioassay, 2,4,6-trichlorophenol has been found to be carcinogenic in mice and rats. Oral doses of 2,4,5-TCP caused liver and kidney lesions in rats and rabbits. Technical grade 2,4,5-TCP is sometimes contaminated with the highly toxic polychlorinated dibenzo-p-dioxins, which may significantly increase the toxicity of the material.

CAS Number: 95-95-4

Chemical Formula: $C_6H_2Cl_3OH$

IUPAC Name: 2,4,5-Trichlorophenol

Important Synonyms and Trade Names: Dowicide 2, Dowicide B, Preventol I

Chemical and Physical Properties

Molecular Weight: 197.5

Boiling Point: 253°C (sublimes)

Melting Point: 68-70.5°C

Specific Gravity: 1.678 at 25°C

Solubility in Water: 1,2000 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, organic solvents, and ligroin

Log Octanol/Water Partition Coefficient: 3.7

Vapor Pressure: 1 mm Hg at 72.0°C

pKa: 7.0

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Transport and Fate

There is very little information available on the environmental transport and fate of 2,4,5-trichlorophenol; however, there is some information concerning 2,4,6-trichlorophenol which may act in a similar fashion. 2,4,6-Trichlorophenol has a low vapor pressure (1 mm Hg at 76.5°C), like 2,4,5-trichlorophenol, and is unlikely to volatilize from water. Photooxidation of 2,4,6-trichlorophenol occurs in the presence of an electron acceptor, and 2,6-dichlorobenzoquinone and 2,6-dichlorohydroquinone are formed. Microbial degradation of 2,4,6-trichlorophenol has been reported. In a number of soil samples, complete degradation of the compound occurred in 1 to 9 days and microbial action in acclimated sludge completely degraded the compound in 5 days. However, there is a report indicating that 2,4,5-trichlorophenol is resistent to degradation by certain soil microbes, possibly because of the meta-substituted chlorine atom in this molecule. Thus, the fate of 2,4,5-trichlorophenol in soil may differ from that of 2,4,6-trichlorophenol.

Health Effects

Although 2,4,5-trichlorophenol has not been tested for carcinogenicity, the NCI bioassay on 2,4,6-trichlorophenol was positive in both rats and mice. 2,4,5-Trichlorophenol gave negative results in the Ames mutagenicity assay, but has been found to promote the formation of papillomas on the skin of mice pretreated with the initiator dimethylbenzanthracene.

McCollister et al. (1961) conducted a number of acute and subchronic studies of 2,4,5-trichlorophenol toxicity in rats and rabbits. The acute oral LD_{50} value of 2,4,5-trichlorophenol was approximately 3,000 mg/kg in the rat. Rats treated 18 times over 24 days with doses ranging from 30 to 1,000 mg/kg did not show adverse effects. Rats supplied diets that gave daily doses of 300 and 1,000 mg of 2,4,5-trichlorophenol per kilogram body weight for 98 days did show liver and kidney effects which were dose related. Rats given lower doses did not show any compound related effects. Rabbits given oral doses of 10 to 500 mg/kg for 28 days exhibited slight renal lesions and, at 500 mg/kg, liver lesions.

In vitro studies on the effect of 2,4,5-trichlorophenol on mitochondrial oxidative phosphorylation showed that it caused complete uncoupling. The concentration of 2,4,5-trichlrophenol that produced a 50% inhibition of ATP production in isolated mitochondria was 6 times less than the concentration of 2,4,6-trichlorophenol.

When considering the health effects of 2,4,5-trichlorophenol, it must be remembered that the technical grade compound is contaminated with polychlorinated dibenzo-p-dioxins, including 2,3,7,8-tetrachlorodibenzo-p-dioxin which is highly toxic and produces a large number of health effects in experimental animals.

Toxicity to Wildlife and Domestic Animals

2,4,5-Trichlorophenol was found to be acutely toxic to the saltwater aquatic species mysid shrimp and sheepshead minnow after a 96-hour exposure to concentrations of 3,830 and 1,660 µg/liter, respectively. No chronic toxicity information on aquatic organisms was available for 2,4,5-trichlorophenol.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Health criterion: 2.6 mg/liter
Organoleptic criterion: 1.0 µg/liter

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WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

2,4,5-TRICHLOROPHENOXYACETIC ACID

Summary

2,4,5-Trichlorophenoxyacetic acid (2,4,5-T) is one of the major constituents of Agent Orange, the major defoliant used in Vietnam. It is commonly contaminated with TCDD, an extremely toxic material that may be responsible for some of the effects associated with exposure to technical 2,4,5-T. These effects include chloracne and the induction of microsomal mixed function oxidase activity. Administration of purified 2,4,5-T has been shown to cause fetal loss, disrupt fetal development, and induce fetal malformations.

CAS Number: 93-76-5

Chemical Formula: $Cl_3C_6H_2OCH_2COOH$

IUPAC Name: 2,4,5-Trichlorophenoxyacetic acid

Important Synonyms and Trade Names: Brushtox, Ded-weed Brush-Killer, 2,4,5-T, Weedar

Chemical and Physical Properties

Molecular Weight: 255.48

Melting Point: 153°C

Solubility in Water: 250 mg/liter

Solubility in Organics: Soluble in alcohol

Vapor Pressure: Less than 8.4×10^{-6} mm Hg at 25°C

Vapor Density: 8.83

pKa: 2.84

Transport and Fate

Photodecomposition of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) in water can occur by a number of different mechanisms. These include photooxidation of the phenoxy side chain and photonucleophilic displacement of Cl by OH to form chlorophenols, and photoreductive dechlorination to form phenoxyacetic acids. Photolysis of 2,4,5-T under dry conditions is also a significant

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environmental fate. Because of its low vapor pressure, volatilization of this compound is not likely to be an important process. At least one experimental study confirmed that volatilization of 2,4,5-T from an aqueous solution is negligible.

2,4,5-T is only weakly adsorbed to soil. In addition, this compound is moderately soluble in water, and experimental studies show that some leaching of 2,4,5-T from soil does occur. This material has been found at low concentrations in groundwater underlying areas to which it has been applied. It has also been detected in the initial rainwater runoff in treated areas. However, most 2,4,5-T remains in the upper layers of soil, and leaching is not thought to be a major transport process.

The environmental persistence of 2,4,5-T is relatively low. For example, 2,4,5-T residues in a forest reportedly declined by 50% in 6 weeks and by 90% in 6 months. Bioaccumulation of 2,4,5-T does not appear to be a significant environmental process.

Health Effects

Currently, there is no conclusive evidence that 2,4,5-T is carcinogenic in humans or experimental animals. Data from studies on experimental animals and in vitro studies suggest that 2,4,5-T is not mutagenic but may damage chromosomes. Administration of 2,4,5-T to pregnant experimental animals disrupts fetal development, causing fetal loss, developmental retardation, and malformations or anomalies. Other acute or chronic effects of 2,4,5-T have not been adequately demonstrated. An oral LD_{50} level of 300 mg/kg is reported for the rat.

The toxic effects of purified 2,4,5-T in experimental animals and humans have not been adequately studied, and other toxic effects observed as a result of exposure to 2,4,5-T formulations, including induction of microsomal mixed function oxidase activity and chloracne, may actually be caused by 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), a common contaminant of these formulations. Information concerning these and other toxic effects is presented in the chemical profile on TCDD.

Toxicity to Wildlife and Domestic Animals

Limited evidence suggests that 2,4,5-T may affect wildlife or domestic animals indirectly by disrupting vegetation density and composition in an area. Herbivores may be affected by changes in the types and amounts of their potential food sources. These changes may favor some species and be detrimental to others. Other animals may lose sources of cover from predators or sites for nest and den building.

Virtually no specific information on the toxicity of 2,4,5-T to wildlife or domestic animals is available. While 2,4,5-T is thought to have relatively low toxicity for vertebrate species, it has been reported that populations of invertebrates, including beneficial insect species, have been adversely affected at field concentrations. Invertebrates may be adversely affected both directly because of the compound's toxicity and indirectly because of the changes 2,4,5-T produces in vegetation growth patterns. Although 2,4,5-T is not reported to have large, direct toxic effects on livestock, there are reports of animal deaths due to alterations in plant chemistry and palatability after 2,4,5-T treatment.

Information on the effects of 2,4,5-T on aquatic species is also limited. Among fish, the LD_{50} value for perch is 55 mg/liter; for guppies, 8 mg/liter; and for rainbow trout, 1.3 mg/liter.

Regulations and Standards

OSHA Standard (air): 10 mg/m^3 TWA

ACGIH Threshold Limit Value: 10 mg/m^3 TWA

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2-(2,4,5-TRICHLOROPHENOXY)PROPIONIC ACID

Summary

2-(2,4,5-Trichlorophenoxy)propionic acid (2,4,5-TP, Silvex) is a broad spectrum herbicide that is commonly contaminated with TCDD. The toxic effects associated with exposure to 2,4,5-TP are generally considered to be caused by this contaminant. However, pure 2,4,5-TD may have an adverse effect on reproduction that is not attributable to TCDD.

CAS Number: 93-72-1

Chemical Formula: $CL_3C_6H_2OCH(CH_3)COOH$

IUPAC Name: 2(2,4,5-Trichlorophenoxy)propionic acid

Important Synonyms and Trade Names: Silvex, 2,4,5-TCPPA, Fenoprop, Kuran, 2,4,5-TP

Chemical and Physical Properties

Molecular Weight: 269.53

Melting Point: 181.6°C

Specific Gravity: 1.640 at 30°C

Solubility in Water: 180 mg/liter

Solubility in Organics: Soluble in acetone, benzene, carbon tetrachloride, ether, heptane, and methanol

Log Octanol/Water Partition Coefficient: 4 (calculated)

Vapor Pressure: Less than 1 mm Hg at 20°C

Transport and Fate

2(2,4,5-Trichlorophenoxy)propionic acid (2,4,5-TP) is not very soluble in water and has a low vapor pressure. It probably is not readily transported in the environment and is likely to be fairly persistent. However, it may volatilize to some degree because of its high activity coefficient in water. Adsorption to soil and sediments is probably an important fate for 2,4,5-TP. Photooxidation and biodegradation may be the ultimate fate processes in the environment, but neither is expected to occur very quickly (Bailey et al. 1970).

Health Effects

2,4,5-TP is contaminated with small amounts of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), and the toxic effects of 2,4,5-TP are generally believed to be caused by this contamination. There is suggestive evidence, however, that 2,4,5-TP may have adverse effects on reproduction that are not attributable to TCDD. This conclusion is based on a comparison of studies using 2,4,5-TP and other studies using essentially uncontaminated 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Uncontaminated 2,4,5-T and uncontaminated 2,4,5-TP are closely related compounds and are considered to have similar effects (Gehring 1980).

The toxic effects of 2,4,5-TP have not been studied well, but they are probably similar to those caused by 2,4,5-T and low levels of TCDD.

Toxicity to Wildlife and Domestic Animals

The 96-hour LC_{50} values for 2,4,5-TP in rainbow trout and bluegills were 15 and 10 mg/liter, respectively. In 5-day feeding studies, Japanese quail had an LD_{50} value greater than 5,000 ppm of 2,4,5-TP when administered in the diet. Ring-necked pheasants under the same regime had an LC_{50} of about 4,500 ppm. 2,4,5-TP is a broadleaf herbicide.

Regulations and Standards

EPA has banned the use of 2,4,5-TP on turf and in aquatic systems.

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TRIS(2,3-DIBROMOPROPYL)PHOSPHATE

Summary

tris(2,3-Dibromopropyl)phosphate (TRIS) is probably persistent in the environment. It is carcinogenic; it induces tumors of the forestomach, lung, kidney, and liver in mice and rats after oral administration and tumors of the forestomach, lung, skin, and mouth in mice after dermal application. TRIS is also mutagenic. Dermal application caused testicular atrophy and kidney damage in rabbits. In humans, dermal exposure may produce allergic skin reactions in susceptible individuals.

CAS Number: 126-72-7

Chemical Formula: $(CH_2BrCHBrCH_2O)_3PO$

Important Synonyms and Trade Names: 2,3-Dibromo-1-propanol phosphate; tris(2,3-ibromopropyl) phosphoric acid ester; TRIS

Chemical and Physical Properties

Molecular Weight: 697.7

Melting Point: 5.5°C

Specific Gravity: 2.27 at 25°C

Solubility in Water: Insoluble

Solubility in Organics: Soluble in all proportions in carbon tetrachloride, chloroform, methylene chloride

Vapor Pressure: 0.00019 mm Hg at 25°C

Transport and Fate

The limited information available concerning tris(2,3-dibromopropyl)phosphate (TRIS) suggests that this compound is relatively persistent in the environment. Hydrolysis, oxidation, and photodegradation are not likely to be significant fate processes. Although slow biodegradation of TRIS in raw sewage is reported to occur, it is not thought to be an important environmental process.

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Given its low vapor pressure, volatilization of TRIS and subsequent atmospheric transport is not likely to be a significant process. Because this compound is virtually insoluble in water, adsorption to particulate matter and sediment may be an important environmental transport process. No adequate empirical data concerning the potential for bioconcentration and biomagnification of TRIS are available. However, because this compound has low solubility in water and is readily soluble in organic solvents, these environmental processes are likely to occur to some extent.

Health Effects

There is sufficient evidence that TRIS is carcinogenic in mice and rats, and can produce tumors of the forestomach, lung, kidney, or liver after oral administration (NCI 1978). This compound also produces benign and malignant tumors of the forestomach, lung, skin, and oral cavity after dermal application in mice (Van Duuren et al. 1978, in IARC 1979). TRIS is also mutagenic in a number of test systems. Based on a limited number of experiments, there is no evidence that TRIS is teratogenic in laboratory animals. TRIS is reported to cause testicular atrophy and kidney damage in rabbits after application to the skin for 3 months. This compound has an oral LD_{50} of 5.24 g/kg in rats. TRIS does not appear to present a significant acute toxic hazard in humans. However, ingestion of this compound is reported to cause some abdominal discomfort and gastrointestinal irritation. Dermal exposure may produce allergic contact sensitization in some subjects.

Toxicity to Wildlife and Domestic Animals

Practically no information concerning the toxicity of TRIS to wildlife and domestic animals exists. Exposure to concentrations of 1 mg/liter caused 50% mortality to goldfish within 4 days (Gutenmann and Lisk 1975) in one study. Central nervous system effects were seen in the fish prior to death. Using a model based on the solubility of selected chemicals in water, a biomagnification potential (concentration in fish/concentration in water) of 338 has been calculated for TRIS.

Regulations and Standards

The manufacture and use of the flame retardant tris(2,3-dibromopropyl)phosphate has been banned in the United States.

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VANADIUM

Summary

Occupational exposure to airborne vanadium has been shown to irritate the skin, eyes, and respiratory tract and to cause bronchitis, bronchospasms, and chest pain. Oral exposure has been associated with gastrointestinal disturbances and discoloration of the oral mucosa. Chronic exposure to vanadium may have an adverse effect on various enzyme systems.

Background Information

Vanadium can exist in the 0, +2, +3, +4, and +5 oxidation states. Elemental vanadium is insoluble in water. Vanadium usually occurs in some oxidized form, and soluble and insoluble vanadium compounds can occur. Vanadium can bind covalently to organic molecules to yield organometallic compounds.

CAS Number: 7440-62-2

Chemical Formula: V

IUPAC Name: Vanadium

Chemical and Physical Properties

Atomic Weight: 50.9

Boiling Point: 3,380°C

Melting Point: 1,890°C

Specific Gravity: 5.96

Solubility in Water: Insoluble

Transport and Fate

The extent to which vanadium is transported in aqueous media is largely determined by the chemical species present and by environmental factors determining its solubility and binding to organic materials. Some vanadium compounds are volatile, and atmospheric transport of fumes as well as particulates can occur. Some bioaccumulation of vanadium occurs. However, in mammals, it appears that excess vanadium can be rapidly excreted in the urine. In humans, it is excreted as sodium metavanadate or ammonium vanadyl tartiate.

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Health Effects

There are no data available to suggest that vanadium has carcinogenic, mutagenic, teratogenic, or reproductive effects in humans or experimental animals. Occupational exposure to airborne vanadium compounds can produce eye and skin irritation. Oral exposure may produce gastrointestinal disturbances and discoloration of the oral mucosa and tongue. There is no evidence of chronic oral toxicity. The most important toxic effects of vanadium are associated with inhalation exposure. Symptoms include acute upper and lower respiratory irritation with mucous discharge and bronchitis, cough, bronchospasm, and chest pain. Acute effects are reported to occur at concentrationos as low as 0.1 mg/m^3. Effects on various enzyme systems may also occur, especially after chronic exposure.

Vanadium is toxic to experimental animals by all routes of administration. Its toxicity generally increases with valence number. The pentavalent chemical forms, such as vanadium pentoxide and the vanadates are the most toxic compounds. In albino mice, an oral LD$_{50}$ of 130 mg/kg vanadium trioxide is reported; a value of 23 mg/kg is reported for vanadium pentoxide and vanadium trichloride.

Toxicity to Wildlife and Domestic Animals

Only limited information was available on the toxicity of vanadium to aquatic organisms (EA 1985). Freshwater fish had 96-hour LC$_{50}$ values ranging from 5,000 to 100,000 μg/liter and generally around 10,000 μg/liter. Daphnids were the only invertebrates studied; a 96-hour LC$_{50}$ value of les than 0.16 μg/lite was reported. Chronic toxicity (5 to 28 day LC$_{50}$ values) was generally seen at around 2,000 μg/liter; the lowest value reported was 500 μg/liter for a 6-day LC$_{50}$ value in the guppy.

Adequate data are not available for characterization of toxicity to wildlife and domestic animals. Calcium vanadate was fatal to a group of chicks fed a diet containing 200 to 600 ppm for 11 to 32 days.

Regulations and Standards

NIOSH Recommended Standards: 1 mg/m^3 TWA
0.05 mg/m^3 Ceiling Level

ACGIH Threshold Limit Value:

0.05 mg/m^3 (vanadium pentoxide, respirable dust and fume)

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VINYL CHLORIDE

Summary

Vinyl chloride is a human carcinogen that causes angiosarcomas of the liver and tumors of the brain, lung, and hemolymphopoietic system. There is suggestive evidence that vinyl chloride has teratogenic and reproductive effects in both humans and animals. Chronic human exposure to vinyl chloride is associated with multiple systemic disorders, including a sclerotic syndrome, acro-osteolysis, and liver damage. Acute human exposure to high concentrations can cause narcosis, respiratory tract irritation, bronchitis, and memory disturbances. Chronic exposure by animals can result in lesions of the liver, kidneys, spleen, and lungs.

CAS Number: 75-01-4

Chemical Formula: CH_2CHCl

IUPAC Name: Chloroethene

Important Synonyms and Trade Names: Chloroethylene, VC, monochloroethylene

Chemical and Physical Properties

Molecular Weight: 62.5

Boiling Point: -13.37°C

Melting Point: -153.8°C

Specific Gravity: 0.9106 at 20°C

Solubility in Water: 1,100 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol ether and carbon tetrachloride

Log Octanol/Water Partition Coefficient: 1.4 (estimated)

Vapor Pressure: 2,660 mm Hg at 25°C

Vapor Density: 2.15

Flash Point: -77.8°C

Transport and Fate

Volatilization from aquatic and terrestrial systems is the most important transport process for distribution of vinyl chloride throughout the environment. Half-lives in aquatic systems range from several minutes to a few hours, depending on temperature, water turbulence, and mixing efficiency. Photooxidation in the troposphere is the dominant environmental fate of vinyl chloride. Vinyl chloride reacts rapidly with hydroxyl radicals, forming hydrogen chloride or formyl chloride. Formyl chloride, if formed, rapidly decomposes to yield carbon monoxide and hydrogen chloride. Vinyl chloride in the atmosphere is expected to be destroyed within one or two days of its release. The hydrogen chloride formed is reported to be removed from the troposphere during precipitation.

Photolysis does not appear to be an important fate process in aquatic systems. Furthermore, photooxidation destroys vinyl chloride before it can reach the stratosphere, where direct photolysis could occur. Based on available information, hydrolysis, sorption, bioaccumulation, and biodegradation do not appear to be important environmental fate processes.

Health Effects

IARC considers vinyl chloride to be a Category I human carcinogen, causing angiosarcomas of the liver and tumors of the brain, lung, and hemolymphopoietic system in humans. Vinyl chloride is carcinogenic in mice, rats, and hamsters; it produces tumors at several sites, including angiosarcomas of the liver, after oral or inhalation exposure. Vinyl chloride, both as a vapor and in solution, is mutagenic in several biological assay systems. In addition, chromosome aberrations including fragments, dicentics and rings, breaks, and gaps have been found in workers occupationally exposed to vinyl chloride. The evidence on its teratogenic and reproductive effects is equivocal. Minor skeletal abnormalities and increased fetal death rates have been observed in the offspring of experimental animals exposed by inhalation to vinyl chloride. In humans, a significant increase in fetal deaths was seen in women whose husbands were exposed to vinyl chloride. Also, an excess number of central nervous system disorders and deformities of the upper alimentary tract, genital organs, and feet were observed in stillborn and live children born in cities with vinyl chloride facilities. However, further research is necessary before the link between vinyl chloride and these observed effects can be positively established.

Acute occupational exposure to high concentrations of vinyl chloride can produce symptoms of narcosis in humans. Respiratory tract irritation, bronchitis, headache, irritaoccur. Chronic exposure to vinyl chloride is associated with multiple systemic disorders, including a sclerotic syndrome, acro-osteolysis, thrombocytopenia, and liver damage, consisting of damage to parenchymal cells, fibrosis of the liver capsule, periportal fibrosis associated with hepatomegaly, and splenomegaly. Concentrations encountered by workers in industries using or producing vinyl chloride are reportedly quite variable and may range from less than the limit of detection to several grams per cubic meter.

Acute inhalation exposure of experimental animals to high concentrations of vinyl chloride can result in narcosis and death. The 2-hour LC_{50} value for rats is 390 g/m^3. Chronic exposure of experimental animals can result in growth disturbances and histopathological and histochemical lesions in the liver, kidneys, spleen, and lungs.

Toxicity to Wildlife and Domestic Animals

No information is available concerning the toxicity of vinyl chloride to domestic animals or wildlife.

Regulation and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of vinyl chloride in water are:

Risk	Concentration
10^{-5}	20 μg/liter
10^{-6}	2.0 μg/liter
10^{-7}	0.2 μg/liter

CAG Unit Risk (USEPA): 1.75×10^{-2} $(mg/kg/day)^{-1}$

OSHA Standards: 26 mg/m^3 TWA
13 mg/m^3/15 min Ceiling Level

ACGIH Threshold Limit Value: Human carcinogen 10 mg/m^3

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XYLENES

Summary

Xylene has been shown to be fetotoxic in rats and mice. In humans, exposure to high concentrations of xylene adversely affects the central nervous system and irritates the mucous membranes.

Background Information

Xylene has three isomers, o-, m-, and p-xylene. These three generally have similar chemical and biological characteristics and therefore will be discussed together.

CAS Number:		
	Mixed:	1330-20-7
	m-Xylene:	108-38-3
	o-Xylene:	95-47-6
	p-Xylene:	106-42-3

Chemical Formula: $C_6H_4(CH_3)_2$

IUPAC Name: Dimethylbenzene

Important Synonyms and Trade Names:

Mixed xylene:	Dimethylbenzene, xylol
m-Xylene:	1,3-Dimethylbenzene, m-xylol
o-Xylene:	1,2-Dimethylbenzene, o-xylol
p-Xylene:	1,4-Dimethylbenzene, p-xylol

Chemical and Physical Properties

Molecular Weight: 106.17

Boiling Point:		
	Mixed:	137-140°C
	m-Xylene:	139°C
	o-Xylene:	144°C
	p-Xylene:	138°C

Melting Point:		
	m-Xylene:	-48°C
	o-Xylene:	-25°C
	p-Xylene:	13°C

Specific Gravity: 0.86

Solubility in Water: 160 mg/liter at 25°C

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Solubility in Organics: Soluble in alcohol, ether, and other organic solvents

Log Octanol/Water Partition Coefficient: 3

Vapor Pressure: 10 mm Hg at 25°C

Vapor Density: 3.7

Flash Point: 25°C (closed cup)

Transport and Fate

Volatilization and subsequent photooxidation by reaction with hydroxyl radicals in the atmosphere are probably important transport and fate processes for xylene in the upper layer of soil and in aquatic environments. Products of the hydroxylation reaction include carbon dioxide, peroxyacetylnitrate (PAN), and cresol. Xylene binds to sediment in water and to organics in soils and undergoes microbial degradation. Biodegradation is probably the most important fate process in both soils and the aquatic environment. Xylenes have been shown to persist for up to 6 months in soil. Because of their low water solubility and rapid biodegradation, xylenes are unlikely to leach into groundwater in high concentrations.

Health Effects

The National Toxicology Program (NTP) is testing xylene for carcinogenicity by administering it orally to rats and mice. Although the results have not been finalized, it does not appear to be carcinogenic in rats.[1] Results have not been reported for mice. Xylene was not found to be mutagenic in a battery of short-term assays. Xylene is not teratogenic but has caused fetotoxicity in rats and mice. Acute exposure to rather high levels of xylene affects the central nervous system and irritates the mucous membranes. There is limited evidence of effects on other organ systems, but it was not possible to attribute these effects solely to xylene as other solvents were present. The oral LD_{50} value of xylene in rats is 5,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

Xylene adversely affected adult trout at concentrations as low as 3.6 mg/liter in a continuous flow system and trout fry avoided xylene at concentrations greater than 0.1 mg/liter. The LC_{50} value in adult trout was determined to be 13.5 mg/liter. LC_{50} values for other freshwater fish were around 30 mg/liter in a static system, which probably underestimated toxicity. Only a few studies have been done on the toxicity of xylene to saltwater species. These indicated that the m- and o-xylene isomers probably have similar toxicities and are probably less toxic than p-xylene, and that saltwater species are generally more susceptible than freshwater species to the detrimental effects of xylene (LC_{50} = 10 mg/liter for m- and o-xylene and LC_{50} = 2 mg/liter for p-xylene). However, it should be stressed that these generalizations are based on limited data.

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[1] W.C. Eastin, NTP Chemical Manager; personal communication, 1984.

No information on the toxicity of xylenes to terrestrial wildlife and domestic animals was available in the literature reviewed. However, because of the low acute toxicity of xylenes it is unlikely that they would be toxic to wild or domestic birds and mammals.

Regulations and Standards

NIOSH Recommended Standards (air): 435 mg/m^3 TWA
870 mg/m^3 10-min Ceiling Level

OSHA Standard (air): 435 mg/m^3 TWA

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ZINC

Summary

Ingestion of excessive amounts of zinc can cause fever, vomiting, and stomach cramps. Zinc oxide fumes can cause metal fume fever. Inhalation of mists or fumes may irritate the respiratory tract, and contact with zinc chloride may irritate the eyes and skin. High levels of zinc in the diet have been shown to retard growth and produce defective mineralization of bone.

Background Information

Zinc generally exists in nature as a salt with a valence of +2, although it is also found in four other stable valences.

CAS Number: 7440-66-6

Chemical Formula: Zn

IUPAC Name: Zinc

Chemical and Physical Properties

Atomic Weight: 65.38

Boiling Point: 907°C

Melting Point: 419.58°C

Specific Gravity: 7.133 at 25°C

Solubility in Water: Insoluble; some salts are soluble

Solubility in Organics: Soluble in acid and alkali

Vapor Pressure: 1 mm Hg at 487°C

Transport and Fate

Zinc can occur in both suspended and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Suspended (undissolved) zinc may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The predominant fate of zinc in aerobic aquatic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their compositions and concentrations; the pH and salinity of the water; the concentrations of complexing ligands; and the concentration of zinc. Concentrations of zinc in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of zinc sulfide limits the mobility of zinc. However, under aerobic conditions, precipitation of zinc compounds is probably important only where zinc is present in high concentrations. Zinc tends to be more readily sorbed at higher pH than lower pH and tends to be desorbed from sediments as salinity increases. Compounds of zinc with the common ligands of surface waters are soluble in most neutral and acidic solutions, so that zinc is readily transported in most unpolluted, relatively organic-free waters.

The relative mobility of zinc in soil is determined by the same factors affecting its transport in aquatic systems. Atmospheric transport of zinc is also possible. However, except near sources such as smelters, zinc concentrations in air are relatively low and fairly constant.

Since it is an essential nutrient, zinc is strongly bioaccumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to be biomagnified. Although zinc is actively bioaccumulated in aquatic systems, the biota appear to represent a relatively minor sink compared to the sediments. Zinc is one of the most important metals in biological systems. Since it is actively bioaccumulated, the environmental concentrations of zinc probably exhibit seasonal fluctuations.

Health Effects

Testicular tumors have been produced in rats and chickens when zinc salts are injected intratesticularly, but not when other routes of administration are used. Zinc may be indirectly important with regard to cancer since its presence seems to be necessary for the growth of tumors. Laboratory studies suggest that although zinc-deficient animals may be more susceptible to chemical induction of cancer, tumor growth is slower in these animals. There is no evidence that zinc deficiency has any etiological role in human cancer. There are no data available to suggest that zinc is mutagenic or teratogenic in animals or humans.

Zinc is an essential trace element that is involved in enzyme functions, protein synthesis, and carbohydrate metabolism. Ingestion of excessive amounts of zinc may cause fever, vomiting, stomach cramps, and diarrhea. Fumes of freshly formed zinc oxide can penetrate deep into the alveoli and cause metal fume fever. Zinc oxide dust does not produce this disorder. Contact with zinc chloride can cause skin and eye irritation. Inhalation of mists or fumes may irritate the respiratory and gastrointestinal tracts. Zinc in excess of 0.25% in the diet of rats causes growth retardation, hypochromic anemia, and defective mineralization of bone. No zinc toxicity is observed at dietary levels below 0.25%.

Studies with animals and humans indicate that metabolic changes may occur due to the interaction of zinc and other metals in the diet. Exposure to cadmium can cause changes in the distribution of zinc, with increases in the liver and kidneys, organs where cadmium also accumulates. Excessive intake of zinc may cause copper deficiencies and result in anemia. Interaction of zinc with iron or lead may also lead to changes that are not produced when the metals are ingested individually.

Toxicity to Wildlife and Domestic Animals

Zinc produces acute toxicity in freshwater organisms over a range of concentrations from 90 to 58,100 µg/liter and appears to be less toxic in harder water. Acute toxicity is similar for freshwater fish and invertebrates. Chronic toxicity values range from 47 to 852 µg/liter and appear to be relatively unaffected by hardness. A final acute-chronic ratio for freshwater species of 3.0 has been reported. Although most freshwater plants appear to be insensitive to zinc, one species, the alga Selenastrum capricornutum, exhibited toxic effects at concentrations from 30 to 700 µg/liter. Reported acute toxicity values range from 2,730 to 83,000 µg/liter for saltwater fish and from 166 to 55,000 µg/liter for invertebrate saltwater species. Zinc produces chronic toxicity in the mysid shrimp at 166 µg/liter. The final acute-chronic ratio for saltwater species is 3.0. Toxic effects are observed in saltwater plant species at zinc concentrations of 50 to 25,000 µg/liter. Bioconcentration factors of edible portions of aquatic organisms range from 43 for the soft-shell clam to 16,700 for the oyster.

Zinc poisoning has occurred in cattle. In one outbreak, poisoning was caused by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited severe enteritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. Zinc concentrations in the liver were extremely high. Based on relatively limited data, some researchers have speculated that exposure to excessive amounts of zinc may constitute a hazard to horses. Laboratory studies and findin in foals living near lead-zinc smelters suggest that excessiv exposure to zinc may produce bone changes, joint afflictions, and lameness. In pigs given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/k deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints. High concentrations of zinc were found in the liver.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(0.83[\ln(\text{hardness})] + 1.95)}$ μg/lite
Chronic toxicity: 47 μg/liter

Saltwater

Acute toxicity: 170 μg/liter
Chronic toxicity: 58 μg/liter

Human Health

Organoleptic criterion: 5 mg/liter

Secondary Drinking Water Standard: 5 mg/liter

NIOSH Recommended Standard: 5 mg/m^3 (zinc oxide)

OSHA Standard: 5 mg/m^3 TWA (zinc oxide)

ACGIH Threshold Limit Values:

Zinc chloride fume:	1 mg/m^3 TWA
	2 mg/m^3 STEL
Zinc oxide fume:	5 mg/m^3 TWA
	10 mg/m^3 STEL
Zinc oxide dust:	10 mg/m^3 TWA (nuisance particulate)
Zinc stearate:	10 mg/m^3 TWA (nuisance particulate)
	20 mg/m^3 STEL

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CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Acenaphthene	Acenaphthene
Acenaphthylene	Acenaphthylene
Acetic acid	Acetic acid
Acetic ether	Ethyl acetate
Acetone	Acetone
Acetylene tetrachloride	1,1,2,2-Tetrachloroethane
trans-Acetylene dichloride	1,2-trans-Dichloroethylene
Acrolein	Acrolein
Acrylaldehyde	Acrolein
Acrylonitrile	Acrylonitrile
Acrylic aldehyde	Acrolein
Agrotect	2,4-Dichlorophenoxyacetic acid
Akar	Chlorobenzilate
Aldrin	Aldrin/Dieldrin
Alkanes	Alkanes
Alkyl benzenes	Alkyl benzenes
Allylaldehyde	Acrolein
2-Aminoethanol	Ethanolamine
Anthracene	Anthracene
Antimony	Antimony
Antimony trioxide	Antimony
Aroclor	Polychlorinated biphenyls
Arsenic	Arsenic
Asbestos	Asbestos
Asex	Sodium chlorate
Atlacide	Sodium chlorate
Attac	Toxaphene
Barium	Barium
1,2-Benzanthracene	Benzo(a)anthracene

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Benzabar	2,3,6-Trichlorobenzoic acid
Benzac	2,3,6-Trichlorobenzoic acid
Benzene	Benzene
Benzene chloride	Chlorobenzene
Benzene hexachloride	Hexachlorocyclohexane
Benzene tetrachloride	1,2,4,5-Tetrachlorobenzene
o-Benzenedicarboxylic acid	n-Dioctyl phthalate
Benzidine	Benzidine
Benzilan	Chlorobenzilate
Benzo(a)anthracene	Benzo(a)anthracene
Benz(b)phenanthrene	Benzo(a)anthracene
Benz(a)phenanthrene	Chrysene
2,3-Benzophenanthrene	Benzo(a)anthracene
Benzosulfonaxole	Benzothiazole
Benzothiazole	Benzothiazole
Beryllium	Beryllium
BHC	Hexachlorocyclohexane
Bibenzyl	Diphenylethane
bis(2-Hydroxyethyl)ether	Diethylene glycol
Brushtox	2,4,5-Trichlorophenoxyacetic acid
Butanol	Butanol
Butyl alcohol	Butanol
Butanone	Methyl ethyl ketone
C-46	Hexachlorobutadiene
Cadmium	Cadmium
Camphechlor	Toxaphene

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Carbide 6-12	Ethyl hexanediol
Carbon hexachloride	Hexachloroethane
Carbon tetrachloride	Carbon tetrachloride
Cellulose nitrate	Nitrocellulose
Cellulose tetranitrate	Nitrocellulose
Chlorate of soda	Sodium chlorate
Chlorate salt	Sodium chlorate
Chlordane	Chlordane
Chlorax	Sodium chlorate
Chlorinated camphene	Toxaphene
Chlorine	Chlorine
Chlorobenzene	Chlorobenzene
Chlorobenzilate	Chlorobenzilate
p-Chloro-m-cresol	p-Chloro-m-cresol
Chloroethane	Chloroethane
Chloroethene	Vinyl chloride
1,-Chloro-2-(beta-chloroethoxy)ethane	bis(2-Chloroethyl)ether
bis(2-Chloroethoxy)ethane	bis(2-Chloroethoxy)ethane
bis(2-chloroethyl)ether)	bis(2-chloroethyl)ether
Chloroethylene	Vinyl chloride
Chloroform	Chloroform
3-Chloro-5-hydroxytoluene	p-Chloro-m-cresol
Chloromethane	Methyl chloride
4-Chloro-3-methylphenol	p-Chloro-m-cresol
Chloro-m-nitrobenzene	1-Chloro-3-nitrobenzene
1-Chloro-3-nitrobenzene	1-Chloro-3-nitrobenzene
Chlorophenothane	DDT
Chlorothene	1,1,1-Trichloroethane
Chromic acid	Chromium
Chromium	Chromium

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CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Chrysene	Chrysene
Cmpd 6-12 Insect Repellant	Ethyl hexanediol
Cobalt	Cobalt
Collodon	Nitrocellulose
Copper	Copper
Cresol	Cresol
Cumene	Alkyl benzenes
Cyanide	Cyanide
Cyanoethylene	Acrylonitrile
Cyanuric acid	Cyanuric acid
Cresylic acid	2,4-Dimethylphenol
2,4-D	2,4-Dichlorophenoxyacetic acid
DBCP	Dibromochloropropane
DCB	Dichlorobenzenes
1,1-DCE	1,1-Dichloroethylene
2,4-DCP	2,4-Dichlorophenol
DDD	DDT
DDE	DDT
DDT	DDT
Cresylic acid	Cresol
Weed Brush Killer	2,4,5-Trichlorophenoxy-acetic acid
DEHP	bis(2-Ethylhexyl)phthalate
4,4'-Diaminobiphenyl	Benzidine
Dibenzyl	Diphenylethane
Dibromochloropropane	Dibromochloropropane
2,3-Dibromo-1-propanol phosphate	Tris(2,3-Dibromopropyl) phosphate
1,2-Dichlorobenzene	Dichlorobenzenes

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
1,3-Dichlorobenzene	Dichlorobenzenes
Dichloro-2,2-dichloroethane	1,1,2,2-Tetrachloroethane
Dichlorodiphenyltrichloroethane	DDT
1,1-Dichloroethane	1,1-Dichloroethane
1,2-Dichloroethane	1,2-Dichloroethane
1,1-Dichloroethene	1,1-Dichloroethylene
2,2'-Dichloroethyl ether	bis(2-Chloroethyl)ether
1,1-Dichloroethylene	1,1-Dichloroethylene
1,2-trans-Dichloroethylene	1,2-trans-Dichloroethylene
Dichloromethane	Methylene chloride
2,4-Dichlorophenol	2,4-Dichlorophenol
2,4-Dichlorophenoxyacetic acid	2,4-Dichlorophenoxyacetic acid
1,2-Dichloropropane	1,2-Dichloropropane
1,3-Dichloropropene	1,3-Dichloropropene
1,3-Dichloropropylene	1,3-Dichloropropene
Dicofol	Dicofol
Dicotext	2,4-Dichlorophenoxyacetic acid
Dieldrin	Aldrin/Dieldrin
Diethyl benzene	Alkyl benzenes
1,4-Diethylenedioxyde	1,4-Dioxane
Diethylene glycol	Diethylene glycol
Diethyl oxide	Tetrahydrofuran
Diethyl ester phthalic acid	Diethyl phthalate
Diethyl ether	Ethyl ether
Diethyl oxide	Ethyl ether
Diethyl phthalate	Diethyl phthalate
1,2-Dihydroxy ethane	Ethylene glycol
Diisobutyl ketone	Diisobutyl ketone

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Dimethylaminoethyl methacrylate	Dimethylaminoethyl methacrylate
Dicophane	DDT
2-Dimethylaminoethyl-2-methylpropenoate	Dimethylaminoethyl methacrylate
Dimethylaniline	Dimethylaniline
Dimethylbenzene	Xylenes
2,6-Dimethyl-4-hepatanone	Diisobutyl ketone
Dimethyl ketone	Acetone
2,4-Dimethyl-1-hydroxybenzene	2,4-Dimethylphenol
Dimethylnitrosamine	Dimethylnitrosamine
2,4-Dimethylphenol	2,4-Dimethylphenol
Dimethylphenylamine	Dimethylaniline
n-Dioctyl phthalate	n-Dioctyl phthalate
Dioform	1,2-trans-Dichloroethylene
1,4-Dioxane	1,4-Dioxane
Dioxin	2,3,7,8-tetrachlorodibenzo-p-dioxin
Dioxins	Polychlorinated dibenzo-p-dioxin
Diphenylethane	Diphenylethane
Diphenyl ether	Phenyl ether
Diphenyl oxide	Phenyl ether
Di(2-ethylhexyl)phthalate	bis(2-Ethylhexyl)phthalate
DMN	Dimethylnitrosamine
DMNA	Dimethylnitrosamine
Dolen	Hexachlorobutadiene
DOP	n-Dioctyl phthalate
Dowicide B	2,4,5-Trichlorophenol
Dowicide 7	Pentachlorophenol
Durene	Alkyl benzenes
Durotox	Pentachlorophenol

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Endrin	Endrin
Endrex	Endrin
1,2-Ethanediol	Ethylene glycol
Ethane trichloride	1,1,2-Trichloroethane
Ethanol	Ethanol
Ethanolamine	Ethanolamine
bis(2-chloroethyl) Ether	bis(2-chloroethyl) Ether
Ethoxyethane	Ethyl ether
Ethyl acetate	Ethyl acetate
Ethyl alcohol	Ethanol
Ethylbenzene	Ethyl benzene
Ethylbenzol	Ethyl benzene
Ethyl chloride	Chloroethane
Ethyl-4,4-dichlorobenzilate	Chlorobenzilate
Ethylene alcohol	Ethylene glycol
Ethylene dichloride	1,2-Dichloroethane
Ethylene diglycol	Ethylene glycol
Ethylene glycol	Ethylene glycol
Ethylene hexachloride	Hexachloroethane
Ethylene trichloride	Trichloroethylene
Ethyl ethanoate	Ethyl acetate
Ethyl ether	Ethyl ether
Ethyl hexanediol	Ethyl hexanediol
Ethyl hexylene glycol	Ethyl hexanediol
Ethyl methyl ketone	Methyl ethyl ketone
bis(2-ethylhexyl)phthalate	bis(2-ethylhexyl)phthalate
di(2-ethylhexyl)phthalate	bis(2-ethylhexyl)phthalate
Ethylidine chloride	1,1-Dichloroethane
Ethylidene dichloride	1,1-Dichloroethane
2-Ethyl-3-propyl-1,3-propanediol	Ethyl hexanediol

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CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Fenoprop	2,4,5-Trichlorophenoxy acetic acid
Fluoranthene	Fluoranthene
Fluorocarbon 11	Trichlorofluoromethane
Fluorotrichloromethane	Trichlorofluoromethane
Folbex	Chlorobenzilate
Formaldehyde	Formaldehyde
Formalin	Formaldehyde
Freon-11	Trichlorofluoromethane
Fumazone	Dibromochloropropane
Gesarol	DDT
Glycol	Ethylene glycol
Glycol Dichloride	1,2-Dichloroethane
Glycol ethylene ether	1,4-Dioxane
Grain alcohol	Ethanol
HCB	Hexachlorobenzene
HCBD	Hexachlorobutadiene
HCH	Hexachlorocyclohexane
Hemimellitine	Alkyl benzenes
Heptachlor	Heptachlor
Heptane	Alkanes
Hexachlorobenzene	Hexachlorobenzene
Hexachlorobutadiene	Hexachlorobutadiene
Hexachlorocyclohexane	Hexachlorocyclohexane
Hexachloroethane	Hexachloroethane
Hexachloroethylene	Hexachloroethane
Hexachlorophene	Hexachlorophene
Hexane	Hexane
Hexide	Hexachlorophene
Hexone	Methyl isobutyl ketone

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
beta-Hydroxyethylamine	Ethanolamine
2-Hydroxynitrolbenzene	2-Nitrophenol
Iron	Iron
Isobutanol	Isobutyl alcohol
Isobutyl alcohol	Isobutyl alcohol
Isobutyl ketone	Diisobutyl ketone
Isobutyl methyl ketone	Methyl isobutyl ketone
Isocyanuric acid	Cyanuric acid
Isodurene	Alkyl benzenes
2-Isopropoxypropane	Isopropyl ether
Isopropyl acetone	Methyl isobutyl ketone
Isopropyl benzene	Alkyl benzenes
Isopropyl ether	Isopropyl ether
Kanechlor	Polychlorinated biphenyls
Kelthane	Dicofol
Klorex	Sodium chlorate
Kuran	2,4,5-Trichlorophenoxy acetic acid
Kusatol	Sodium chlorate
Lead	Lead
Lindane	Hexachlorocyclohexane
Lithium	Lithium
Magnesium	Magnesium
Manganese	Manganese
m-chloronitrobenzene	1-Chloro-3-nitrobenzene
MEK	Methyl ethyl ketone
Mendrin	Endrin
Mercury	Mercury
Mesitylene	Alkyl benzenes
Metaphor	Methyl parathion
Methacrylate	Methacrylic acid

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CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Methacrylic acid	Methacrylic acid
Methacrylic acid, methyl ester	Methacrylic acid
Methanal	Formaldehyde
Methanecarboxylic acid	Acetic acid
Methane dichloride	Methylene chloride
Methanol	Methanol
Methylacrylic acid	Methacrylic acid
Methyl alcohol	Methanol
Methyl benzene	Toluene
Methyl chloride	Methyl chloride
Methyl chloroform	1,1,1-Trichloroethane
2-Methyl dodecane	Alkanes
3-Methyl hexane	Alkanes
2-Methyl pentane	Alkanes
3-Methyl pentane	Alkanes
2-Methyl tetradecane	Alkanes
2-Methyl tridecane	Alkanes
2,2-Methylene-bis(3,4,6-trichlorophenol)	Hexachlorophene
Methylene chloride	Methylene chloride
Methylene dichloride	Methylene chloride
Methyl ethyl benzene	Ethyl toluene
Methyl ethyl ketone	Methyl ethyl ketone
Methyl isobutyl ketone	Methyl isobutyl ketone
Methyl methacrylate	Methacryllic acid
Methyl-2-methyl-2-propenoate	Methacryllic acid
Methyl parathion	Methyl parathion
4-Methyl-2-pentanone	Methyl isobutyl ketone

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
2-Methyl propanol	Isobutyl alcohol
2-Methyl-2-propenoate	Methacrylic acid
MIBK	Methyl isobutyl ketone
MIK	Methyl isobutyl ketone
Mitigan	Dicofol
Monochlorobenzene	Chlorobenzene
Monochloroethane	Chloroethane
Monochloroethylene	Vinyl chloride
Monochloromethane	Methyl chloride
Monoethanolamine	Ethanolamine
Moth balls	Naphthalene
Nabac	Hexachlorophene
Naphthalene	Naphthalene
Naphthene	Naphthalene
NDMA	Dimethylnitrosamine
Nemagon	Dibromochloropropane
Neocid	DDT
Nickel	Nickel
Nitrocellulose	Nitrocellulose
Nitrochlorobenzene	1-Chloro-3-nitrobenzene
Nitro cotton	Nitrocellulose
2-Nitrophenol	2-Nitrophenol
Nitrosodimethylamine	Dimethylnitrosamine
n-Methyl-n-nitrosomethanamine	Dimethylnitrosamine
n-Nitrosodimethylamine	Dimethylnitrosamine
N,N-Dimethylbenzeneamine	Dimethylaniline
n,n-dimethylnitrosamine	Dimethylnitrosamine
N-phenyldimethylamine	Dimethylaniline
Octyl phthalate	n-Dioctyl phthalate
Orthophosphoric acid	Phosphoric acid
1,1'-Oxybis(2-chloroethane)	bis(2-Chloroethyl) ether

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CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
2,2-Oxydiethanol	Diethylene glycol
PAH	Polycyclic aromatic hydrocarbons
Paranaphthalene	Anthacene
PCB	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzo-p-dioxins
PCE	Tetrachloroethylene
PCP	Pentachlorophenol
P-Dioxane	1,4-Dioxane
Pegalan	Methacrylic acid
Pentachlorophenol	Pentachlorophenol
Pentadecane	Alkanes
Perchlorobenzene	Hexachlorobenzene
Perchlorobutadiene	Hexachlorobutadiene
Perchloroethylene	Tetrachloroethylene
Perchloromethane	Carbon tetrachloride
Phenacide	Toxaphene
Phenanthrene	Phenanthrene
Phenol	Phenol
Phenol trinitrate	Picric acid
Phenox	2,4-Dichlorophenoxyacetic acid
Phenoxybenzene	Phenyl ether
Phenyl chloride	Chlorobenzene
Phenyldimethylamine	Dimethylaniline
Phenylethane	Alkyl benzenes
Phenyl ether	Phenyl ether
Phenylmethane	Toluene
Phosphoric acid	Phosphoric acid
Phosphorus (white)	Phosphorus (white)

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
bis(2-ethylhexyl) Phthalate	bis(2-ethylhexyl) Phthalate
Picric acid	Picric acid
PNA	Polycyclic aromatic hydrocarbons
Polychlorinated biphenyls	Polychlorinated biphenyls
Polychlorinated dibenzo-p-dioxin	Polychlorinated dibenzo-p-dioxin
Polycyclic aromatic hydrocarbons	Polycyclic aromatic hydrocarbons
Polynuclear aromatic hydrocarbons	Polycyclic aromatic hydrocarbons
Preventol	2,4,5-Trichlorophenol
Propanone	Acetone
2-Propenal	Acrolein
Properenitrile	Acrylonitrile
2-Propenenitrile	Acrylonitrile
2-Propen-1-one	Acrolein
Propyl carbinol	1-Butanol
Propylenechloride	1,2-Dichloropropane
Propylenedichloride	1,2-Dichloropropane
Pseudocumene	Alkyl benzenes
Rat Nip	Phosphorus (white)
Rutgers 6-12	Ethyl hexanediol
Selenium	Selenium
Silver	Silver
Silvex	2,4,5-Trichlorophenoxy propionic acid
Soda chlorate	Sodium chlorate
Sodium	Sodium
Sodium chlorate	Sodium chlorate
Soluble gun cotton	Nitrocellulose

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Stoddard solvent	Stoddard solvent
Strobane-T	Toxaphene
Sym-triazinetriol	Cyanuric acid
2,4,5-T	2,4,5-Trichlorophenoxy-acetic acid
Tar camphor	Naphthalene
2,3,6-TBA	2,3,6-Trichlobenzoic acid
1,1,1-TCA	1,1,1-Trichloroethane
TCB	Trichlorobenzene
TCE	Trichloroethylene
TCDD	2,3,7,8-Tetrachlorodibenzo-p-dioxin
TCPPA	2,4,5-Trichlorophenoxy propionic acid
TEL	Tetraethyl lead
Telone	1,3-Dichloropropene
1,2,4,5-Tetrachlorobenzene	1,2,4,5-Tetrachlorobenzene
2,3,7,8-Tetrachlorodibenzo-p-dioxin	2,3,7,8-Tetrachlorodibenzo-p-dioxin
1,1,2,2-Tetrachloroethane	1,1,2,2-Tetrachloroethane
Tetrachloroethene	Tetrachlorothylene
Tetrachlorothylene	Tetrachlorothylene
Tetrachloromethane	Carbon tetrachloride
Tetradioxin	2,3,7,8-Tetrachlorodibenzo-p-dioxin
Tetraethyl lead	Tetraethyl lead
Tetraethyl plumbane	Tetraethyl lead
Tetrahydrofuran	Tetrahydrofuran
Tetramethyl benzene	Alkyl benzenes
Tetramethylene oxide	Tetrahydrofuran
Thallium	Thallium

CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
1-Thia-3-azaindene	Benzothiazole
Titanium	Titanium
Toluene	Toluene
Toluol	Toluene
Toxaphene	Toxaphene
2,4,5-TP	2,4,5-Trichlorophenoxy propionic acid
1,3,5-Triazine-2,4,6 (1H,3H,5H)-trione	Cyanuric acid
Trichlorobenzene	Trichlorobenzene
2,3,6-Trichlorobenzoic acid	2,3,6-Trichlorobenzoic acid
1,1,1-Trichloroethane	1,1,1-Trichloroethane
1,1,2-Trichloroethane	1,1,2-Trichloroethane
Trichloroethene	Trichloroethylene
Trichloroethylene	Trichloroethylene
Trichlorofluoromethane	Trichlorofluoromethane
Trichloromethane	Chloroform
2,4,5-Trichlorophenol	2,4,5-Trichlorophenol
2,4,5-Trichlorophenoxyacetic acid	2,4,5-Trichlorophenoxy acetic acid
2,4,5-Trichlorophenoxy propionic acid	2,4,5-Trichlorophenoxy propionic acid
Tricresol	Cresol
Tricyanic acid	Cyanuric acid
Triethylene glycol dichloride	bis(2-Chloroethoxy)ethane
Triglycol dichloride	bis(2-Chloroethoxy)ethane
Trihydroxycyanidine	Cyanuric acid
2,4,6-Trihydroxyl-1,3,5-triazine	Cyanuric acid

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CHEMICAL NAME/ SYNONYM/TRADE NAME	LISTED UNDER THE FOLLOWING CHEMICAL NAME
Trimethyl benzene	Alkyl benzenes
2,4,6-Trinitrophenol	Picric acid
TRIS	Tris(2,3-Dibromopropyl) phosphate
Tris(2,3-Dibromopropyl) phosphate	Tris(2,3-Dibromopropyl) phosphate
Tris(2,3-ibromopropyl) phosphoric acid ester	Tris(2,3-dibromopropyl) phosphate
Trysben	2,3,6-Trichlorobenzoic acid
Undecane	Alkanes
Vanadium	Vanadium
Vinegar acid	Acetic acid
Vinegar naphtha	Ethyl acetate
VC	Vinyl chloride
VDC	1,1-Dichloroethylene
Vinyl chloride	Vinyl chloride
Weedar	2,4,5-Trichlorophenoxy-acetic acid
Yellow Phosphorus	Phosphorus (white)
Vinyl cyanide	Acrylonitrile
Vinylidene chloride	1,1-Dichloroethylene
Vinyl trichloride	1,1,2-Trichloroethane
Wofatox	Methyl parathion
Wood alcohol	Methanol
Xylene	Xylene
2,4-Xylenol	2,4-Dimethylphenol
Xylol	Xylene
Zinc	Zinc

APPENDIX A

HAZARD CRITERIA

It is usually difficult to give a simple yes or no answer to the question of whether or not a chemical is hazardous because in most cases, gradations exist in such things as the quality of data or species-specificity of the effects. In addition, the degree of hazard posed by a chemical depends on the dose or environmental concentration and on the duration and other circumstances of exposure. In order to provide a dichotomous response in these cases, it is necessary to determine general criteria for classifying chemicals as having specific toxicity. The criteria used in this system for determining whether a chemical poses a particular type of hazard are outlined below.

Carcinogenicity

A compound is classified as a carcinogenic if it is a known or suspected human carcinogen, if it has been shown to be carcinogenic at a particular site in more than one species or sex in an animal bioassay, or if it has been shown to increase the incidence of site-specific malignant tumors in a single species or sex, and there is a statistically significant dose-response relationship in more than one exposed group. Observations such as site of application tumors in a skin painting study or sporadic significant results in a bioassay will not be considered indications of carcinogenicity unless supported by other evidence.

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Reproductive Toxicity/Teratogenicity

Chemicals are classified as teratogens and reproductive toxins if there is suggestive evidence of an effect in humans or if at least one study in whole animals is clearly positive. Unsupported in vitro evidence is considered sufficient to classify a chemical as a reproductive toxicity/teratogenicity hazard.

Mutagenicity

A chemical is classified as mutagenic if it has given a positive result in at least one of the mammalian in vivo or bacterial or mammalian cell in vitro assays for mutagenicity. However, negative studies will be considered, and in some cases may outweigh a weak positive response.

Acute Toxicity

A compound will be considered to be acutely toxic if it has an oral $LD_{50} \leq 100$ mg/kg, an inhalation $LC_{50} \leq 400$ mg/m^3, or a dermal $LD_{50} \leq 400$ mg/kg.

Chronic Toxicity

Chemicals will be considered to cause chronic toxicity if they cause serious irreversible effects other than cancer or reproductive effects after extended exposure to oral doses of less than 100 mg/kg/day, inhalation concentrations less than 400 mg/m^3, or dermal doses less than 400 mg/kg/day.

Domestic Animal Toxicity

A chemical will be considered to be toxic to domestic animals if a demonstrated serious toxic effect has been seen in the field. Also, chemicals that cause reproductive toxicity, teratogenicity, or subchronic toxicity at oral doses of less than 100 mg/kg/day will be considered as domestic animal hazards unless they are unlikely to be present at toxic levels off site.

Environmental Toxicity

A chemical is classified as hazardous to aquatic wildlife if an acute LC_{50} value in aquatic organisms is less than 1000 μg/liter, or if the chemical has chronic effects at less than 100 μg/liter.

A chemical is classified as hazardous to terrestrial wildlife if toxicity has been seen in the field or if the chemical is acutely toxic or causes reproductive toxicity/teratogenicity to representative species at oral doses less than 100 mg/kg body weight.

Chemicals that are persistent in the environment and that are toxic at levels up to 10 times less than those indicated above, are also classified as hazardous to the environment.

HAZARD CLASSIFICATION

Chemical	CAS Number	Hazard						
		Carcino-genicity	Reproductive/ Teratogenicity	Mutagen-icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environ-mental Toxicity
Acenaphthene	83-32-9							
Acenaphthylene	208-96-8							
Acetic acid	64-19-7							
Acetone	67-64-1							
Acrolein	107-02-8				X			X
Acrylonitrile	107-13-1	X	X	X	X			
Aldrin	309-00-2	X	X	X	X	X	X	X
Alkanes								
Alkyl benzenes								
Anthracene	120-12-7			X				
Antimony	7440-36-0		X	X		X		
Arsenic	7440-38-2	X	X	X		X	X	X
Asbestos	1332-21-4	X				X		
Barium	7440-39-3		X		X			

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Chemical	CAS Number	Hazard Carcino- genicity	Reproductive/ Teratogenicity	Mutagen- icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Envi menta Toxic
Benzene	71-43-2	X	X	X		X		
Benzidine	92-87-5	X		X		X		
Benzo(a)anthracene	56-55-3	X		X				
Benzo(a)perylene	191-24-2			X				
Benzo(a)pyrene	50-32-8	X	X	X				
Benzo(k)fluoranthene	207-08-9							
Benzothiazole	95-16-9							
Beryllium	7440-41-7	X				X		X
gamma-BHC (lindane)	58-89-9	X	X	X		X		X
1-Butanol	71-36-3							
di-n-Butyl phthalate	84-74-2		X			X		
Cadmium	7440-43-9	X	X			X	X	X
Carbon tetrachloride	56-23-5	X				X		
Chlordane	57-74-9	X	X	X		X		X
Chlorine	7782-50-5				X			X

		Hazard						
Chemical	CAS Number	Carcinogenicity	Reproductive/ Teratogenicity	Mutagenicity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environmental Toxicity
Chlorobenzene	108-90-7							
Chlorobenzilate	510-15-6	X						
Chloroethane	75-00-3							
bis(2-Chloroethoxy)ethane	112-26-5							
bis(2-Chloroethyl)ether	111-44-4	X			X			
Chloroform	67-66-3	X						
p-Chloro-m-cresol	59-50-7					X		
1-Chloro-3-nitrobenzene	121-73-3							X
Chromic acid	7738-94-5			X				
Chromium	7440-47-3	X	X	X		X		X
Chrysene	218-01-9	X		X				
Cobalt	7440-48-4							
Copper	7440-50-8							X
Cresol	1319-77-3							
Cyanide	57-12-5				X		X	X
Cyanuric acid	108-80-5							

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Chemical	CAS Number	Hazard						
		Carcino-genicity	Reproductive/ Teratogenicity	Mutagen-icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environ mental Toxicit
1,3-Dichloropropene	542-75-6			X				
Dicofol	115-32-2	X	X					
Dieldrin	60-57-1	X	X	X				X
Diethylphthalate	84-66-2		X	X				
Diisobutyl ketone	108-83-8							
Dimethylaminoethyl methacrylate	2439-35-2							
Dimethylaniline	121-69-7							
Dimethylnitrosamine	62-75-9	X	X	X	X			
2,4-Dimethylphenol	105-67-9							
n-Dioctyl phthalate	117-84-0		X					
1,4-Dioxane	123-91-1	X		X				
Diphenylethane	103-29-7							
Endrin	72-20-8		X		X	X	X	X
Ethanol	64-17-5		X	X				
Ethanolamine	141-43-5							

		Hazard						
Chemical	CAS Number	Carcino-genicity	Reproductive/ Teratogenicity	Mutagen-icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environ-mental Toxicit
DBCP (Dibromochloropropane)	96-12-18	X	X	X				
p,p'-DDE	72-55-9	X	X			X	X	X
p,p'-DDD	72-54-8	X	X			X	X	X
o,p'-DDD	53-19-0	X	X			X	X	X
DDT (p,p')	50-29-3	X	X			X	X	X
o,p'-DDT	789-02-6	X	X			X	X	X
Dibenzo(a,h)anthracene	53-70-3	X		X				
Dichlorobenzene	95-50-1							
1,1-Dichloroethane	75-34-3							
1,2-Dichloroethane	107-06-2	X		X		X		
1,1-Dichloroethylene	75-35-4	X	X	X				
1,2-trans-Dichloroethylene	156-60-5							
2,4-Dichlorophenol	120-83-2							X
2,4-Dichlorophenoxy acetic acid	94-75-7		X	X				
1,2-Dichloropropane	78-87-5	X		X				

Chemical	CAS Number	Hazard: Carcino-genicity	Hazard: Reproductive/ Teratogenicity	Hazard: Mutagen-icity	Hazard: Acute Toxicity	Hazard: Chronic Effect	Hazard: Domestic Animal Toxicity	Hazard: Environ-mental Toxicity
Ethyl acetate	141-78-6							
Ethylbenzene	100-41-4		x					
Ethylene glycol	107-21-1		x	x				
Ethyl ether	60-29-7		x					
Ethyl hexanediol	94-96-2							
bis-2-Ethylhexyl phthalate	117-81-7	x	x					
Fluoranthene	206-44-0							
Fluorene	86-73-7							
Formaldehyde	50-00-0	x		x	x	x		
Heptachlor	76-44-8	x	x	x	x	x	x	x
Hexachlorobenzene	118-74-1	x	x			x	x	x
Hexachlorobutadiene	87-68-3	x		x		x		x
Hexachlorocyclohexane	608-73-1	x				x		x
Hexachloroethane	67-72-1	x						
Hexachlorophene	70-30-4		x		x			
Hexane[2]	110-54-3		x			x		

		Hazard						
Chemical	CAS Number	Carcino-genicity	Reproductive/ Teratogenicity	Mutagen-icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environ-mental Toxicity
Indeno(1,2,3-cd) pyrene	193-39-5	x		x				
Iron	7439-89-6							
Isobutyl alcohol	78-83-1			x				
Isopropyl ether	108-20-3							
Lead	7439-92-1		x			x	x	x
Lithium	7439-93-2		x		x			
Magnesium	7439-95-4							
Manganese	7439-96-5			x				
Mercury	7439-97-6		x	x	x	x	x	x
Methacrylic acid	79-41-4			x				
Methanol	67-56-1			x				
Methyl chloride	74-87-3	x	x	x		x		
Methylene chloride	75-09-2	x		x				
Methyl ethyl ketone	78-93-3		x					
Methyl isobutyl ketone	108-10-1							

Chemical	CAS Number	Hazard						
		Carcino-genicity	Reproductive/ Teratogenicity	Mutagen-icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environ mental Toxicit
Methyl methacrylate	80-62-6		x	x				
Methyl parathion	298-00-0		x		x			x
Naphthalene	91-20-3					x		
Nickel	7440-02-0	x	x			x		x
Nitrocellulose	9004-70-0							
2-Nitrophenol	88-75-5							
4-Nitrophenol	100-02-7							
Pentachlorophenol	87-86-5		x					x
Phenanthrene	85-01-8				x			
Phenol	108-95-2							
Phenyl ether	101-84-8							
Phosphoric acid	7664-38-2							
Phosphorus (white)	7723-14-0		x		x	x	x	x
Picric acid	88-89-1			x				
Polychlorinated biphenyls	1336-36-3	x	x					x

		Hazard						
Chemical	CAS Number	Carcinogenicity	Reproductive/ Teratogenicity	Mutagenicity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environmental Toxicity
Polychlorinated dibenzo-p-dioxins		x	x		x	x	x	x
Pyrene	129-00-0							
Selenium	7782-49-2		x		x		x	x
Silver	7440-22-4				x			
Sodium (metal)	7440-23-5							
Sodium chlorate	7775-09-9			x				
Stoddard solvent	8052-41-3							
Sulfuric acid	7664-93-9							
1,2,4,5-Tetrachlorobenzene	95-94-3							
2,3,7,8-Tetrachloro-dibenzo-p-dioxin	1746-01-6	x	x		x	x	x	x
1,1,2,2-Tetrachloroethane	79-34-5	x	x	x		x		
Tetrachloroethylene	127-18-4	x	x	x				
Tetraethyl lead	78-00-2				x	x		x

		Hazard						
Chemical	CAS Number	Carcino-genicity	Reproductive/ Teratogenicity	Mutagen-icity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Enviro mental Toxici
Tetrahydrofuran	109-99-9							
Thallium	7440-28-0				X		X	X
Titanium	7440-32-6							
Toluene	108-88-3		X					X
Toxaphene	8001-35-2	X	X		X		X	X
Trichlorobenzene	50-31-7							
1,1,1-Trichloroethane	71-55-6			X				
1,1,2-Trichloroethane	79-00-5	X						
Trichloroethylene	79-01-6	X		X				
Trichlorofluoromethane	75-69-4							
2,4,5-Trichlorophenoxy acetic acid	93-76-5		X					
2,4,5-Trichlorophenoxy propionic acid	93-72-1		X					X
2,4,5-Trichlorophenol	95-95-4							X
tris(2,3-Dibromopropyl) phosphate	126-72-7	X		X				

		Hazard						
Chemical	CAS Number	Carcinogenicity	Reproductive/ Teratogenicity	Mutagenicity	Acute Toxicity	Chronic Effect	Domestic Animal Toxicity	Environmental Toxicit
Vanadium	7440-62-2				X			
Vinyl chloride	75-01-4	X	X	X		X		
Xylene	1330-20-7		X					
Zinc	7440-66-6						X	X

APPENDIX B

CAG UNIT RISK

The Carcinogen Assessment Group (CAG) of EPA has performed quantitative risk assessments on numerous chemicals. These assessments were performed using data from the best available studies at the time the assessment was performed; the data were fitted to the particular mathematical model considered most appropriate. A "unit risk," defined as the lifetime cancer risk to humans associated with continuous exposure to a unit dose of 1 mg/kg/day, was calculated. The 95th percentile upper confidence limit for unit risk is given in the following table as a slope. This value can be divided into the generally acceptable lifetime risk of 10^{-6} to determine the daily dose in mg/kg/day associated with this risk level. It should be stressed that the data used to generate the unit risk numbers and the methods of extrapolation are relatively inexact and utilize conservative assumptions; therefore, the unit risk values should only be considered as at best, order of magnitude approximations of the upper limit on potential risk. This is reflected in the last column of the following table, which indicates the potency of each chemical to order of magnitude on a logarithmic scale. On this scale, a chemical with an index of +6 is about one million (10^6) times more potent than a chemical with an index of zero.

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RELATIVE CARCINOGENIC POTENCIES AMONG 54 CHEMICALS EVALUATED BY THE CARCINOGEN ASSESSMENT GROUP AS SUSPECT HUMAN CARCINOGENS

Compounds	CAS Number	Level of evidence[a]		Grouping based on IARC criteria	Slope[b] $(mg/kg/day)^{-1}$	Molecular weight	Potency index[c]	Order of magnitude ($\log_{10}$ index)
		Humans	Animals					
Acrylonitrile	107-13-1	L	S	2A	0.24(W)	53.1	$1x10^{+1}$	+1
Aflatoxin B_1	1162-65-8	L	S	2A	2900	312.3	$9x10^{+5}$	+6
Aldrin	309-00-2	I	L	2B	11.4	369.4	$4x10^{+3}$	+4
Allyl chloride	107-05-1				$1.19x10^{-2}$	76.5	$9x10^{-1}$	0
Arsenic	7440-38-2	S	I	1	15(H)	149.8	$2x10^{+3}$	+3
B[a]P	50-32-8	I	S	2B	11.5	252.3	$3x10^{+3}$	+3
Benzene	71-43-2	S	S	1	$2.9x10^{-2}$(W)	78	$2x10^{0}$	0
Benzidene	92-87-5	S	S	1	234(W)	184.2	$4x10^{+4}$	+5
Beryllium	7440-41-7	L	S	2A	2.6	9	$2x10^{+1}$	+1
1,3-Butadiene	106-99-0	I	S	2B	$1.0x10^{-1}$(I)	54.1	$5x10^{0}$	+1
Cadmium	7440-43-9	L	S	2A	6.1(W)	112.4	$7x10^{+2}$	+3
Carbon tetrachloride	56-23-5	I	S	2B	$1.30x10^{-1}$	153.8	$2x10^{+1}$	+1
Chlordane	57-74-9	I	L	3	1.61	409.8	$7x10^{+2}$	+3

(continued on the following page)

Compounds	CAS Number	Level of evidence[a] Humans	Level of evidence[a] Animals	Grouping based on IARC criteria	Slope[b] (mg/kg/day)$^{-1}$	Molecular weight	Potency index[c]	Order of magnitude (log$_{10}$ index)
Chlorinated ethanes								
1,2-Dichloroethane	107-06-2	I	S	2B	$9.1x10^{-2}$	98.9	$9x10^{0}$	+1
hexachloroethane	67-72-1	I	L	3	$1.42x10^{-2}$	236.7	$3x10^{0}$	0
1,1,2,2-Tetrachloroethane	79-34-5	I	L	3	0.20	167.9	$3x10^{+1}$	+1
1,1,2-Trichloroethane	79-00-5	I	L	3	$5.73x10^{-2}$	133.4	$8x10^{0}$	+1
Chloroform	67-66-3	I	S	2B	$7x10^{-2}$	119.4	$8x10^{0}$	+1
Chromium VI	7440-47-3	S	S	1	41(W)	100	$4x10^{+3}$	+4
DDT	50-29-3	I	S	2B	0.34	354.5	$1x10^{+2}$	+2
Dichlorobenzidine	91-94-1	I	S	2B	1.69	253.1	$4x10^{+2}$	+3
1,1-Dichloroethylene (Vinylidene chloride)	75-35-4	I	L	3	1.16(I)	97	$1x10^{+2}$	+2
Dichloromethane (Methylene chloride)	75-09-2	I	L	3	$6.3x10^{-4}$(I)	84.9	$5x10^{-2}$	-1
Dieldrin	60-57-1	I	S	2B	30.4	380.9	$1x10^{+4}$	+4
2,4-Dinitrotoluene	121-14-2	I	S	2B	0.31	182	$6x10^{+1}$	+2
Diphenylhydrazine	122-66-7	I	S	2B	0.77	180	$1x10^{+2}$	+2
Epichlorohydrin	106-89-8	I	S	2B	$9.9x10^{-3}$	92.5	$9x10^{-1}$	0
Bis(2-chloroethyl)ether	111-44-4	I	S	2B	1.14	143	$2x10^{+2}$	+2

(continued on the following page)

Compounds	CAS Number	Level of evidence[a] Humans	Level of evidence[a] Animals	Grouping based on IARC criteria	Slope[b] $(mg/kg/day)^{-1}$	Molecular weight	Potency index[c]	Order of magnitude ($\log_{10}$ index)
Bis(chloromethyl)ether	542-88-1	S	S	1	9300(I)	115	$1x10^{+6}$	+6
Ethylene dibromide (EDB)	106-93-4	I	S	2B	41	187.9	$8x10^{+3}$	+4
Ethylene oxide	75-21-8	L	S	2A	$3.5x10^{-1}$(I)	44.1	$2x10^{+1}$	+1
Heptachlor	76-44-8	I	S	2B	3.37	373.3	$1x10^{+3}$	+3
Hexachlorobenzene	118-74-1	I	S	2B	1.67	284.4	$5x10^{+2}$	+3
Hexachlorobutadiene	87-68-3	I	L	3	$7.75x10^{-2}$	261	$2x10^{+1}$	+1
Hexachlorocyclohexane								
technical grade					4.75	290.9	$1x10^{+3}$	+3
alpha isomer	319-84-6	I	S	2B	11.12	290.9	$3x10^{+3}$	+3
beta isomer	319-85-7	I	L	3	1.84	290.9	$5x10^{+2}$	+3
gamma isomer	58-89-9	I	L	2B	1.33	290.9	$4x10^{+2}$	+3
Hexachlorodibenzodioxin	34465-46-8	I	S	2B	$6.2x10^{+3}$	391	$2x10^{+6}$	+6
Nickel	7440-02-0	L	S	2A	1.15(W)	58.7	$7x10^{+1}$	+2
Nitrosamines								
Dimethylnitrosamine	62-75-9	I	S	2B	25.9(not by q_1^*)	74.1	$2x10^{+3}$	+3
Diethylnitrosamine	55-18-5	I	S	2B	43.5(not by q_1^*)	102.1	$4x10^{+3}$	+4
Dibutylnitrosamine	924-16-3	I	S	2B	5.43	158.2	$9x10^{+2}$	+3
N-nitrosopyrrolidine	930-55-2	I	S	2B	2.13	100.2	$2x10^{+2}$	+2
N-nitroso-N-ethylurea	759-73-9	I	S	2B	32.9	117.1	$4x10^{+3}$	+4

(continued on the following page)

Compounds	CAS Number	Level of evidence[a]		Grouping based on IARC criteria	Slope[b] $(mg/kg/day)^{-1}$	Molecular weight	Potency index[c]	Order of magnitude ($\log_{10}$ index)
		Humans	Animals					
N-nitroso-N-methylurea	684-93-5	I	S	2B	302.6	103.1	$3x10^{+4}$	+4
N-nitroso-diphenylamine	86-30-6	I	S	2B	$4.92x10^{-3}$	198	$1x10^{0}$	0
PCBs	1336-36-3	I	S	2B	4.34	324	$1x10^{+3}$	+3
Phenols								
2,4,6-Trichlorophenol	88-06-2	I	S	2B	$1.99x10^{-2}$	197.4	$4x10^{0}$	+1
Tetrachlorodibenzo-p-dioxin (TCDD)	1746-01-6	I	S	2B	$1.56x10^{+5}$	322	$5x10^{+7}$	+8
Tetrachloroethylene	127-18-4	I	L	3	$5.1x10^{-2}$	165.8	$8x10^{0}$	+1
Toxaphene	8001-35-2	I	S	2B	1.13	414	$5x10^{+2}$	+3
Trichloroethylene	79-01-6	I	L/S	3/2B	$1.1x10^{-2}$	131.4	$1x10^{0}$	0
Vinyl chloride	75-01-4	S	S	1	$1.75x10^{-2}$(I)	62.5	$1x10^{0}$	0

[a] S = Sufficient evidence; L = Limited evidence; I = Inadequate evidence.

[b] Animal slopes are 95% upper-bound slopes based on the linearized multistage model. They are calculated based on animal oral studies, except for those indicated by I (animal inhalation), W (human occupational exposure), and H (human drinking water exposure). Human slopes are point estimates based on the linear nonthreshold model. Not all of the carcinogenic potencies presented in this table represent the same degree of certainty. All are subject to change as new evidence becomes available. The slope value is an upper bound in the sense that the true value (which is unknown) is not likely to exceed the upper bound and may be much lower, with a lower bound approaching zero. Thus, the use of the slope estimate in risk evaluations requires an appreciation for the implication of the upper bound concept as well as the "weight of evidence" for the likelihood that the substance is a human carcinogen.

[c] The potency index is a rounded-off slope in $(mmol/kg/day)^{-1}$ and is calculated by multiplying the slopes in $(mg/kg/day)^{-1}$ by the molecular weight of the compound.

Source: U.S. Environmental Protection Agency (USEPA). 198
Health Assessment Document for Chloroform. Office
of Health and Environmental Assessment, Washington